TO STUDY THE CLINICAL PROFILE OF LIVER DISEASE IN PREGNANT FEMALES ADMITTED IN HOSPITAL & ITS EFFECT ON MATERNO-FETAL OUTCOME

A THESIS SUBMITTED TO BABA FARID UNIVERSITY OF

HEALTH SCIENCES, FARIDKOT (PUNJAB)

IN THE PARTIAL FULFILLMENT

FOR THE AWARD OF DEGREE OF

DOCTOR OF MEDICINE

IN

GENERAL MEDICINE

2024

Submitted By

DR. ARSHDEEP KAUR

DEPARTMENT OF GENERAL MEDICINE
DAYANAND MEDICAL COLLEGE AND HOSPITAL
LUDHIANA, PUNJAB

DECLARATION BY THE CANDIDATE

I hereby declare that the thesis entitled **"TO STUDY THE CLINICAL PROFILE OF LIVER DISEASE IN PREGNANT FEMALES ADMITTED IN HOSPITAL & ITS EFFECT ON MATERNO-FETAL OUTCOME"** submitted towards partial fulfillment of the requirements for the award of MD IN **GENERAL MEDICINE** degree to BFUHS is a compilation of original work carried out by me under the supervision of **Dr. Sandeep Puri,** Former Principal & Professor, Department of General Medicine, **Dr. Suman Puri**, Professor & Unit Head, Department of Obstetrics & Gynaecology and **Dr. Omesh Goyal**, Professor, Department of Gastroenterology. No part of this thesis has formed the basis for the award of any degree previously.

_____

DR. ARSHDEEP KAUR
Department of General Medicine
Dayanand Medical College & Hospital,
Ludhiana, Punjab

Date:

Place:

CERTIFICATE OF SUPERVISORS

This is to certify that the work incorporated in this thesis entitled **"TO STUDY THE CLINICAL PROFILE OF LIVER DISEASE IN PREGNANT FEMALES ADMITTED IN HOSPITAL & ITS EFFECT ON MATERNO-FETAL OUTCOME"** has been carried out by **Dr. Arshdeep Kaur** under our direct supervision. The data incorporated in this thesis is original and genuine and has been carried out by the candidate herself.

Signature of Supervisor

Dr. Sandeep Puri
Former Principal & Professor
Department of General Medicine
DMCH, Ludhiana

Signature of Co-Supervisors

Dr. Suman Puri
Professor & Unit Head
Department of Obs. & Gynae.
DMCH, Ludhiana

Dr. Omesh Goyal
Professor
Department of Gastroenterology
DMCH, Ludhiana

Date:

ENDORSEMENT BY THE HEAD OF THE INSTITUTE

This is to certify that the thesis entitled **"TO STUDY THE CLINICAL PROFILE OF LIVER DISEASE IN PREGNANT FEMALES ADMITTED IN HOSPITAL & ITS EFFECT ON MATERNO-FETAL OUTCOME"** is a bonafide research work done by **Dr. Arshdeep Kaur** under the supervision of **Dr. Sandeep Puri,** Former Principal & Professor, Department of General Medicine, **Dr. Suman Puri**, Professor & Unit Head, Department of Obstetrics & Gynaecology and **Dr. Omesh Goyal**, Professor, Department of Gastroenterology, in this institution.

Principal

Dr. G S Wander
Principal
DMC&H, Ludhiana

Date:

COPYRIGHT

Declaration by the Candidate

I hereby declare that the Baba Farid University of Health Sciences, Faridkot, shall have the rights to preserve & use this thesis in print or electronic format for academic/study purpose by the research scholar/students.

Signature

Dr. Arshdeep Kaur
Department of General Medicine
Dayanand Medical College & Hospital,
Ludhiana, Punjab

Date:

© Baba Farid University of Health Sciences, Faridkot

Dedicated

to

my Family

&

Teachers

ACKNOWLEDGEMENT

I would like to express my heartfelt gratitude to everyone who had supported and guided me throughout the course of my thesis. Their contributions have been invaluable and deeply appreciated.

First and foremost, I would like to thank the Almighty Waheguru who has always showered his blessings on me. His presence i feel at each and every step of my life. He's the one who holds my hand and guides me through everything.

Words are less to express my gratitude to my Parents, ***Mr.Jasminder Singh Grover*** *and* ***Mrs Daljit Kaur*** *for their love, support and selfless sacrifices that have made me whatever I am today. I owe my perseverance and determination to the values you have instilled in me. Thank you for your patience, understanding, and encouragement throughout this journey.*

I want to express my gratitude to my Brother, ***Mr. Pavneet Singh*** *Grover, who has been my pillar of strength throughout this journey. Your encouragement has been a constant source of strength and your dedication to my education has made this achievement possible. Without your guidance, this task would have remained incomplete.*

I want to thank my Sister ***Mrs. Sonpreet Kaur****, my Sister in law* ***Mrs. Supreet Kaur*** *and my Brother* ***Mr. Avneet*** *Singh for their care, affection, help and inspiration throughout.*

Much love to my niece ***Harsheen*** *and my nephews* ***Japgun*** *and* ***Gunraz*** *for being my sunshine on a rainy day.*

I express my profound gratitude to my esteemed teacher and mentor, ***Dr. Sandeep Puri****, Former Professor and Principal, Department of Medicine, DMCH. Your expertise, patience, and insightful guidance have been pivotal in shaping this research. I am sincerely grateful for the time and effort you invested in mentoring me and for continuously urging me to pursue excellence. I feel privileged and honoured to work under such an illustrious guide and teacher.*

I am deeply thankful to my respected co-supervisors, ***Dr. Suman Puri****, Professor, Department of Obstetrics and Gynaecology and* ***Dr. Omesh Goyal****, Professor, Department of Gastroenterology at DMCH, for their invaluable contribution, continual assistance, and guidance throughout this journey.*

I would like to thank my teachers ***Dr.Harmeet pal Singh Dhooria*** *and* ***Dr.Davinderpal Singh Dhanota*** *for their motivation and encouragement in completing this uphill task.*

I would like to thank my colleagues, friends and juniors for helping me in periods of distress throughout my post-graduation period. Special thanks to my friend ***Dr. Ajaypal*** *for invaluable support day in, day out for the entire course of work.*

Thanks to all the patients who have been part of this study and the entire technical staff of DMCH who provided me with all the valuable help.

A Special Thanks to ***Mr. Vikas Sharma, Mr.Avinash, Mr. Sushil*** *and* ***Mrs. Namita****, Research and Development centre for their efforts.*

Dr. Arshdeep Kaur

LIST OF ABBREVIATIONS

AFLP	- ACUTE FATTY LIVER OF PREGNANCY
ALF	-ACUTE LIVER FAILURE
ACLF	-ACUTE ON CHRONIC LIVER FAILURE
APH	-ANTEPARTUM HAEMORRHAGE
ALT	-ALANINE AMINOTRANSFERASE
AST	-ASPARTATE AMINOTRANSFERASE
CLD	-CHRONIC LIVER DISEASE
CS	-CESAREAN SECTION
CT	-COMPUTED TOMOGRAPHY
DIC	-DISSEMINATED INTRAVASCULAR COAGULATION
FGR	-FETAL GROWTH RESTRICTION
HELLP	-HAEMOLYSIS, ELEVATED LIVER ENZYMES, LOW PLATELET COUNTS
HAV	-HEPATITIS A VIRUS
HBV	-HEPATITIS B VIRUS
HCV	-HEPATITIS C VIRUS
HDV	- HEPATITIS D VIRUS
HEV	- HEPATITIS E VIRUS
HG	-HYPEREMESIS GRAVIDARUM
ICP	-INTRAHEPATIC CHOLESTASIS OF PREGNANCY
ICU	-INTENSIVE CARE UNIT
INR	**-**INTERNATIONAL NORMALIZED RATIO
IUD	-INTRAUTERINE DEATH
LCFAD	-LONG-CHAIN FATTY ACID DEHYDROGENASE
LFT	-LIVER FUNCTION TEST
LSCS	-LOWER SEGMENT CESAREAN SECTION
NAFLD	-NON ALCOHOLIC FATTY LIVER DISEASE
NICU	- NEONATAL INTENSIVE CARE UNIT
NVD	-NORMAL VAGINAL DELIVERY
SD	-STANDARD DEVIATION
UDCA	-URSODEOXYCHOLIC ACID
USG	-ULTRASONOGRAPHY

ABSTRACT

Introduction: Liver diseases during pregnancy pose significant challenges for gynecologists and hepatologists, with a 3% incidence rate among all pregnancies, leading to materno-fetal morbidity and mortality. Liver diseases during pregnancy often goes undetected and undiagnosed because LFTs are not typically part of routine antenatal tests, so LFTs should be done routinely in pregnant females as a screening tool.

Aims and Objectives: 1) To study the Clinical profile of liver disease in pregnant females admitted in hospital.

2) To study and compare the Materno-fetal outcome among hospitalized pregnant females with or without liver disease

Materials and Methods :A prospective study was conducted in 100 cases of pregnant females with liver disease and 50 controls pregnant females without liver disease admitted in department of Medicine and Gynaecology in a tertiary care hospital, Dayanand Medical College and Hospital, Ludhiana during a 15 month period from 15th september,2022 to 15th December,2023**.** Liver function tests were studied and compared between the two groups. Total numbers of pregnancies were noted, out of which females fulfilling the criteria were considered for the study.

Results: The obeservations from our study were as follows: In our study,there were 280 cases of deranged liver function tests (LFTs) out of total 1480 deliveries during a 15-month period. From these, 100 patients and 50 controls that met the inclusion criteria were included in the study.The incidence of liver disorder during pregnancy was found to be 18% in our study. Out of 100 patients,62 patients had pregnancy specific liver disorders,22 patients were found to have liver disorders unrelated to pregnancy and in 16 patients no definitive cause could be determined for the abnormal LFTs.ICP was the most prevalent disorder among pregnancy specific liver disorder accounting for 45 cases out of 62 (72%),followed by HELLP syndrome 6 cases out of 62 (9.6%).Tropical fever was the most common among liver disorders unrelated to pregnancy accounting for 11 cases out of 22 (50%),followed by Hepatitis 5 cases out of 22 (22.7%).In our study, the highest number of patients were within the 26-30 years age group (36%).80% cases presented in the third trimester, with the majority being multigravida (59%).Pain abdomen was the most observed symptom (49%).The most common sign seen was icterus (19%).Mean AST was 274.0 and Mean ALT was 247.1 in study group. C -section was most common mode of delivery in our study (78%) while in control group majority of patients had vaginal delivery (70%).The most common indication for C -section was fetal distress (36%).Mean fetal birth weight was 2.2 kg in study group and 2.8 kg in control group. Among fetal outcome, Out of 100 deliveries in study group,48 babies had normal live birth (48%),While In Control group out of 50, 47 babies had normal live birth (94%).Regarding maternal outcome in study group out of 100 cases-57 females had normal outcome (57%) while in control group out of 50 controls, 49 females had normal outcome (98%).

Conclusion: ICP was the most common pregnancy specific liver disorder in our study.Tropical fever was the most common among liver disorders unrelated to pregnancy. study group 48% babies and 57% females had normal outcome while in control group 94% babies and 98% females had normal outcome respectively. Therefore managing pregnant women with jaundice is crucial because it affects both the mother and the unborn baby. The key to successful management lies in identifying the underlying cause of the jaundice early.

Keywords: Liver disorder,Pregnancy, Jaundice ,Maternal outcome, Fetal outcome

TABLE OF CONTENTS

S.NO.	TOPIC	PAGES
I	INTRODUCTION	1-3
II	AIMS AND OBJECTIVES	4
III	REVIEW OF LITERATURE	5-25
IV	MATERIALS AND METHODS	26-29
V	OBSERVATIONS AND RESULTS	30-64
VI	DISCUSSION	65-74
VII	SUMMARYAND CONCLUSION	75-78
VIII	BIBLIOGRAPHY	79-93

APPENDICES–I

- PROFORMA
- MASTER CHART

APPENDICES–II

- APPROVAL LETTER OF THESIS PLAN
- THESIS PLAN

LIST OF TABLE

Table No.	Title	Page No.
1	PHYSIOLOGICAL CHANGES IN VARIOUS INVESTIGATIONS DURING NORMAL PREGNANCY	2
2	CLASSIFICATION OF HEPATIC DISORDERS OF PREGNANCY	3
3	SOME COMMON CHARACTERSTICS OF COMMON PREGNANCY RELATED LIVER-DISEASES	6-7
4	SWANSEA CRITERIA FOR THE DIAGNOSIS OF ACUTE FATTY LIVER OF PREGNANCY	15
5	THE TENNESSEE AND MISSISSIPPI CLASSIFICATION OF HELLP IS SHOWN IN SUPPLEMENTARY	19
6	DISTRIBUTION OF STUDY GROUP AND CONTROL GROUP ACCORDING TO AGE	30
7	DISTRIBUTION OF STUDY GROUP ACCORDING TO ETIOLOGY	31
8	DISTRIBUTION OF STUDY GROUP AND CONTROL GROUP ACCORDING TO TRIMESTER	33
9	DISTRIBUTION OF STUDY GROUP AND CONTROL GROUP ACCORDING TO GESTATIONAL AGE	35
10	MEAN GESTATIONAL AGE IN OUR STUDY	36
11	DISTRIBUTION OF STUDY GROUP AND CONTROL GROUP ACCORDING TO COMORBIDITIES	38
12	DISTRIBUTION OF STUDY GROUP AND CONTROLGROUP ACCORDING TO GRAVIDA	39
13	DISTRIBUTION OF STUDY GROUP AND CONTROL GROUP ACCORDING TO SYMPTOMS	40
14	DISTRIBUTION OF STUDY GROUP AND CONTROL	42

	GROUP ACCORDING TO SIGNS	
15	MEAN HAEMOGLOBIN (Hb) IN OUR STUDY	43
16	MEAN TLC IN OUR STUDY	44
17	MEAN PLATELET COUNT IN OUR STUDY	45
18	MEAN LIVER ENZYMES IN OUR STUDY	46
19	COMPARISON OF DISEASES IN STUDY GROUP ACCORDING TO MEAN LIVER ENZYMES	47
20	MEAN BILIRUBIN (TOTAL,DIRECT) IN OUR STUDY	48
21	MEAN TOTAL PROTEIN AND ALBUMIN IN OUR STUDY	49
22	MEAN OF BILE ACIDS IN OUR STUDY	50
23	DISTRIBUTION OF STUDY GROUP AND CONTROL GROUP ACCORDING TO MODE OF DELIVERY	50
24	DISTRIBUTION OF STUDY GROUP AND CONTROL GROUP ACCORDING TO INDICATIONS OF CESAREAN SECTION	52
25	DISTRIBUTION OF STUDY GROUP AND CONTROL GROUP ACCORDING TO FETAL GENDER	54
26	DISTRIBUTION OF STUDY GROUP AND CONTROL GROUP ACCORDING TO FETAL BIRTH WEIGHT	55
27	MEAN FETAL BIRTH WEIGHT OF STUDY GROUP AND CONTROL GROUP	56
28	DISTRIBUTION OF STUDY GROUP AND CONTROL GROUP ACCORDING TO FETAL OUTCOME	58
29	.DISTRIBUTION OF STUDY GROUP AND CONTROL GROUP ACCORDING TO FETAL COMPLICATIONS	60

30	DISTRIBUTION OF STUDY GROUP AND CONTROL GROUP ACCORDING TO MATERNAL OUTCOME	61
31	DISTRIBUTION OF STUDY GROUP ACCORDING TO MATERNAL COMPLICATIONS	63

LIST OF FIGURE

FIG. No.	Title	Page No.
1	DISTRIBUTION OF STUDY GROUP AND CONTROL GROUP ACCORDING TO AGE	31
2	DISTRIBUTION OF STUDY GROUP ACCORDING TO ETIOLOGY	32
3	DISTRIBUTION OF STUDY GROUP AND CONTROL GROUP ACCORDING TO TRIMESTER	34
4	DISTRIBUTION OF STUDY GROUP AND CONTROL GROUP ACCORDING TO GESTATIONAL AGE	36
5	MEAN GESTATIONAL AGE IN OUR STUDY	37
6	DISTRIBUTION OF STUDY GROUP AND CONTROL GROUP ACCORDING TO COMORBIDITIES	39
7	DISTRIBUTION OF STUDY GROUP AND CONTROL GROUP ACCORDING TO GRAVIDA	40
8	DISTRIBUTION OF STUDY GROUP AND CONTROL GROUP ACCORDING TO SYMPTOMS	41
9	DISTRIBUTION OF STUDY GROUP AND CONTROL GROUP ACCORDING TO SIGNS	43
10	MEAN HAEMOGLOBIN (Hb) IN OUR STUDY	44
11	MEAN TLC IN OUR STUDY	45
12	MEAN PLATELET COUNT IN OUR STUDY	46
13	MEAN LIVER ENZYMES IN OUR STUDY	47
14	MEAN BILIRUBIN (TOTAL, DIRECT) IN OUR STUDY	48

15	MEAN TOTAL PROTEIN AND ALBUMIN IN OUR STUDY	49
16	DISTRIBUTION OF STUDY GROUP AND CONTROLGROUP ACCORDING TO MODE OF DELIVERY	51
17	DISTRIBUTION OF STUDY GROUP AND CONTROL GROUP ACCORDING TO INDICATIONS OF CESAREAN SECTION	53
18	DISTRIBUTION OF STUDY GROUP AND CONTROL GROUP ACCORDING TO FETAL GENDER	55
19	DISTRIBUTION OF STUDY GROUP AND CONTROL GROUP ACCORDING TO FETAL BIRTH WEIGHT	56
20	MEAN FETAL BIRTH WEIGHT OF STUDY GROUP AND CONTROL GROUP	57
21	DISTRIBUTION OF STUDY GROUP AND CONTROL GROUP ACCORDING TO FETAL OUTCOME	59
22	DISTRIBUTION OF STUDY-GROUP AND CONTROL GROUP ACCORDING TO FETAL COMPLICATIONS	61
23	DISTRIBUTION OF STUDY-GROUP AND CONTROL GROUP ACCORDING TO MATERNAL OUTCOME	62
24	DISTRIBUTION OF STUDY-GROUP ACCORDING TO MATERNAL COMPLICATIONS	64

BABA FARID UNIVERSITY OF HEALTH SCIENCES
Sadiq Road, Faridkot-151203 (Pb.) INDIA
Phone No. 91-1639-256253 and Fax No. 91-1639-256234
Website: www.bfuhs.ac.in, Email: bfuhspc@gmail.com
(BFUHS – UNIVERSITY LIBRARY- Coordinator APC)

11-BFUHS (LI-PC-134)24/696 Dated: July 09, 2024

Plagiarism Verification Certificate

Title of the thesis: **To Study The Clinical Profile Of Liver Disease Among Pregnant Females Admitted In The Hospital & Its Effect On Materno-Fetal Outcome**

Total Page: **78 (Seventy Eight only) eFSPC (eFile Sent for Plagiarism Check)**

Researcher: **Arshdeep Kaur**

Supervisor: **Dr. Sandeep Puri**

Research Centre: **Dayanand Medical College And Hospital, Ludhiana**

This is to report that the above thesis was scanned for similarity detection. Process and outcome is given below:

DID:	**2089782**	Software used:	**DRILLBIT**
Date:	**July 09, 2024**	Similarity Index:	**7% (Below 10 Percent)**

Total words count: **13733 (eFSPC)**

The complete report is submitted for review by the Supervisor and Co-Supervisor.

University Coordinator/
Name & Signature (Checked by)
(From University library)

The complete report of the above thesis has been reviewed by the undersigned. (Check Box)

- ☐ The similarity index is below accepted norms.
- ☐ The similarity index is above accepted norms, because of the following reasons:

1.....
2.....
3…….....
4…….....
5……

The thesis may be considered for the award of degree. (Relevant documents attached).

Research Scholar **Supervisor**

INTRODUCTION

Liver disease during pregnancy is a significant complication, posing challenges for both gynecologists and hepatologists. It affects nearly 3% of pregnancies and affecting materno-fetal health, some of which may be fatal for both.[1–6] Common liver disorders of pregnancy include preeclampsia, HELLP syndrome (hemolysis, elevated liver enzymes, and low platelets), acute fatty liver of pregnancy, hyperemesis gravidarum, and intrahepatic cholestasis of pregnancy. Prompt investigation and management of these conditions are essential to reduce materno-fetal morbidity and mortality.

When diagnosing liver disease in pregnancy, it's important to consider the normal physiological and hormonal changes that occur during this period. These changes can mimic liver disease, complicating diagnosis and management. For instance, the hyperestrogenic state in pregnancy can cause spider angioma and palmar erythema, which are usually associated with cirrhosis. Hemodynamic changes, including increased plasma volume and cardiac output, decreased vascular resistance, and renin-angiotensin-aldosterone system activation, can present or worsen symptoms of portal hypertension.[7] During a normal pregnancy, albumin levels decline by trimester due to hemodilution, and alkaline phosphatase levels increase, especially in the 3rd trimester, due to placental rather than hepatic origin. Serum alanine aminotransferase (ALT) levels typically decrease in the third trimester, while aspartate aminotransferase (AST) levels remain unchanged.[8] Gamma-glutamyl transpeptidase levels decrease by 25%[9] and total serum bile acid concentrations increase postprandially.[10] Pregnancy is a pro-coagulant state, with increases in specific clotting factors and fibrinogen, though prothrombin time (PT) and partial thromboplastin time (pTT) remain normal. Therefore, elevated levels of AST, ALT, total bilirubin, and international normalized ratio during pregnancy warrant evaluation for new or previously undiagnosed liver disease. Any

abnormalities in these parameters may indicate hepatic pathology in pregnancy.

Table 1; Physiological changes in various investigations during normal pregnancy

Test	Normal Range
Bilirubin	Unchanged or slightly decreased
Aminotransferases	Unchanged
Prothrombin time	Unchanged
Alkaline phosphatase	Increases 2 to 4-fold
Fibrinogen	Increases 50%
Globulin	Increases in α and β globulins
α-fetoprotein	Moderate increase, especially with twins
WBC	Increases
Ceruloplasmin	Increases
Cholesterol	Increases 2-fold
Triglycerides	Increases
Globulin	Decreases in gamma-globulin
Hemoglobin	Decrease in later pregnancy

A systematic approach to treatment is necessary, involving a detailed clinical history, physical examination, laboratory tests, and radiographic evaluation. The clinical history should include previous pregnancy-related diseases and liver complications, drug use, transfusions, whether pregnancies were carried to term, and oral contraceptive use. Additionally, the patient should be assessed for symptoms of liver dysfunction, such as nausea, vomiting, jaundice, generalized pruritus, abdominal pain, polyuria, and polydipsia, excluding other chronic conditions like diabetes.[11-14]

Hepatic disorders of pregnancy is categorized into two main groups: those pregnancy related and those unrelated or coincide with pregnancy, including pre-existing chronic liver diseases exacerbated by pregnancy(table 2).[2,6,13] Pregnancy-related liver diseases include intrahepatic cholestasis of pregnancy (ICP), acute fatty liver of pregnancy (AFLP) and HELLP syndrome. Pre-eclampsia (PE) and

hyperemesis gravidarum (HG) are also frequent association with hepatic abnormalities.[4,5,11] Non-pregnancy-related hepatic diseases encompass acute and chronic viral hepatitis, biliary diseases such as primary sclerosing cholangitis and gallstones, cirrhosis with or without portal hypertension, and, vascular alterations like Budd–Chiari syndrome (BCS), metabolic disorders, Wilson's disease, drug-induced hepatotoxicity, autoimmune liver diseases, and post-liver transplantation status (table 2).[2,5,6,15]

Table 2: Classification of hepatic disorders of pregnancy.[2, 15]

Pregnancy-related liver diseases	Pregnancy-unrelated liver diseases	
	Pre-existing	Co-incident with pregnancy
Hyperemesis gravidarum	Hepatitis B, C and E	Acute viral hepatitis
Intrahepatic cholestasis of pregnancy	Cirrhosis and portal hypertension	Biliary diseases (e.g. cholelithiasis, primary sclerosing cholangitis)
Acute fatty liver of pregnancy	Autoimmune liver disease	Vascular alterations (Budd–Chiari syndrome)
Pre-eclampsia and eclampsia	Wilson's disease	Metabolic diseases
HELLP syndrome	Post liver transplantation state	Liver tumors
	Non-alcoholic fatty liver disease	Drug-induced hepatotoxicity

Pregnancy-associated liver disorders often manifest during specific trimesters, notably the 3rd trimester. Among these, pre-eclampsia related disorders are the most prevalent[16], while liver diseases unrelated to pregnancy can emerge at any point during pregnancy. Mortality rates ranging from 0% to 25% have been documented among mothers with pregnancy-related liver diseases.[6,17,18] Prompt identification of clinical symptoms and deranged LFTs(liver function tests) is crucial for accurate diagnosis and management planning. Therapy decisions must consider the materno-fetal well-being, with urgent diagnosis imperative in severe liver conditions as immediate delivery may significantly impact materno-fetal outcomes. Maternal prognosis hinges on factors such as the specific hepatic disorder type, the extent of liver function impairment in synthesis, metabolism, and excretion, as well as gestational age at the time of delivery.[6,12,13].

AIMS AND OBJECTIVES

- To study the Clinical profile of liver disease in pregnant females admitted in hospital.
- To study and compare the Materno-fetal outcome among hospitalized pregnant females with or without liver disease.

REVIEW OF LITERATURE

Liver disorders during pregnancy, although not extensively studied individually, form a varied group of conditions sharing common symptoms but differing in biochemical characteristics. They represent about 3% of all pregnancies and pose a significant diagnostic challenge.[19] Effective management is crucial for the well-being of both mother and fetus, as these conditions can contribute to mortality rates of up to 25%.[19,20] Among these, five disorders are specific to pregnancy: hyperemesis gravidarum (HG), intrahepatic cholestasis of pregnancy (ICP), liver issues linked with preeclampsia such as HELLP syndrome (hemolysis, elevated liver enzymes, and low platelet count), hepatic infarction and rupture, and acute fatty liver of pregnancy (AFLP). Although their prevalence is around 3%[1], it can vary based on geographic and ethnic factors. These disorders significantly impact maternal and fetal health, with maternal mortality rates still falling short of international targets. Trends in the United States indicate a concerning increase in liver disorder-related maternal deaths over recent years.[21]

Utilizing data from the National Inpatient Survey, we found that the hospitalization rate for pregnancy-related liver diseases (including AFLP and ICP) increased from 5.97 to 8.91 per 1000 pregnancy hospitalizations between 2002–2004 and 2008–2010.[22] Diagnostic tests like screening for various viral hepatitis markers (HAV immunoglobulin [Ig]M, anti-HCV, HBsAg, anti-HBc, anti-HDV, anti-HEV IgM), Epstein-Barr virus, cytomegalovirus, and herpes simplex virus, as well as antinuclear antibody, anti-smooth muscle antibody, IgG, ceruloplasmin, drug and alcohol screening including acetaminophen, celiac antibody, and Doppler ultrasound during pregnancy will help in early diagnosis of liver diseases in pregnancy.[23] The normal range of ALT for healthy pregnant women is 6 to 32 U/L. For X times the upper limit of normal determination, the upper limit of normal, ranging from 19 to 25 U/L, is utilized.[23]

Table 3. -Some common characteristics of common Pregnancy related liver-diseases

Disease	Hyperemesis gravidarum	Intrahepatic cholestasis of pregnancy	Acute fatty liver of pregnancy	Pre eclampsia with liver dysfunction	HELLP syndrome
Trimester	First	Second–third	Third-postpartum	> 20 weeks	Third-postpartum (25%)
Frequency	0.3–2%	0.1–5%	0.01%	5–10%	0.2–0.6%
Clinical features	Vomiting, dehydration, weight loss, ketosis	Pruritus, jaundice (25%)	Abdominal pain, vomiting, polydipsia/polyuria, encephalopathy	Abdominal pain, hypertension, proteinuria, headache, blurred vision, peripheral edema	Abdominal pain, vomiting, proteinuria, headache, peripheral edema
Ascites	–	–	+/−	–	−
Platelets	–	–	↓	↓	↓
Hemolysis	–	–	−	↑	+/− ↑
Bilirubin	<4mg/dL	<5mg/dL	<10mg/dL	<5mg/dL	<5mg/dL
Bile acids	–	30–100×	−	–	−
Aminotransferases	1–2× (50%)	1–5×	5–10×	1–100× (20–30%)	1–100×
LDH	–	–	↑	↑	≥600IU/L
ALP	–	↑	↑	↑ mild	↑

Hypoglycemia	–	–	+/−	–	−
Uric acid	–	–	↑ (80%)	↑	↑
Creatinine	–	–	↑	↑	−
Proteinuria	–	–	+/− ↑	↑	+/− ↑
Liver imaging(USG, CT)	Normal parenchyma without biliary obstruction	Should excluded cholelithiasis	Fatty infiltration, bright liver	Hepatic infarcts, hematomas, rupture	Hepatic infarcts, hematomas, rupture
Liver histopathology	–	Hepatocellular bile and canalicular bile plugs, cholestasis without parenchymal inflammation	Microvesicular steatosis, foamy hepatocytes, hepatic necrosis	Fibrin deposition, hemorrhage, hepatocellular necrosis	Fibrin deposition, hemorrhage, hepatocellular necrosis
Complications	Usually resolves by 18 weeks without complications	Late intrauterine death	Maternal and fetal/perinatal mortality. 50% will have evidence of PE	Liver rupture and necrosis. Maternal and fetal/perinatal mortality.	Maternal and fetal/perinatal mortality
Treatment	Supportive, rehydration, antiemetics, vitamins supplementation: B1, B6, B12, C	Ursodeoxycholic acid and delivery at 37 weeks	Delivery at 34 weeks	Control of hypertension and urgent delivery to avoid eclampsia	Delivery at 34 weeks or even before 24–34 weeks given corticoids for fetal lung maturation
Recurrence	+++	+++	Rarely25% risk in defects of fatty acids β oxidation	++	++ 4–19%

Hyperemesis Gravidarum

HG is characterized by severe nausea and persistent vomiting, leading to dehydration, ketosis, and a weight loss > 5% of body weight.[5,6] It affects approximately 0.3–2.0% of pregnancies in the 1st trimester, sometimes necessitating hospitalization. Symptoms typically commence before the ninth week of pregnancy and cease before 20th week.[5,6,12,14] While HG is primarily diagnosed by exclusion, assessment for other possible causes is recommended in atypical patients, particularly if vomiting persists into the second trimester. Although HG is not classified as a true liver disease, around half of patients exhibit abnormal LFTs.[5,6,13,15] Clinical manifestations include dehydration, raised renal function (RFTs) values, metabolic alkalosis, electrolyte imbalances, and erythrocytosis. Mild to moderate elevations in aminotransferases, with jaundice occurring in a minority, are observed in 15-50% of patients. Other biochemical derangements such as raised serum lipase and serum amylase levels may also occur.[5,24,25] Additionally, abdominal ultrasound should reveal normal hepatict issue with no sign of biliary obstruction, however ultrasound is essential as HG could be related with multiple pregnancies and hydatidiform mole. HG risk is higher when there is increased body mass index, pre-existing diabetes, psychiatric disorders, asthma, hyperthyroidism during previous pregnancies, or a history of HG in previous pregnancies.[6,11,13,27]

Maternal and Fetal Outcomes

HG can cause significant maternal morbidity. Medical complications include Wernicke's encephalopathy (as a consequence of thiamine deficiency), gastrointestinal bleeding secondary to trauma, Thromboembolism and electrolyte abnormalities (like hyponatremia or hypokalemia both of which can cause potentially fatal arrhythmia).[28-30] HG is related with raised suicidal ideation rates.[31,32] and 7% of affected women terminate a wanted pregnancy.[33,34] Certain studies have found higher rates of growth restriction in infants [35], however not all of them show this.[2] There is proof that offspring of HG-affected

mothers are more likely to experience neuro developmental delays and autism spectrum disorders in the future.[37,38,39]

Management [24,40-42]

Individualized first-line pharmacological therapy should be provided based on the timing of symptoms and the response to prior treatments. According to guidelines from the UK and Australia, if women are unable to tolerate oral antiemetics, non-oral delivery of antihistamine and phenothiazine therapy is advised initially. According to US standards, doxylamine and vitamin B6 should be taken together. Extra therapy can be added if women only partially respond to first-line medications. These include ondansetron, metoclopramide, and prochlorperazine[43], all of which have strong safety records for use during pregnancy. Women should be informed that extrapyramidal adverse effects are possible with metoclopramide and should cease taking the medication right away if manifesting any such symptoms.

Women with refractory HG who are unable to maintain hydration or weight may require inpatient management and corticosteroid medication. Thiamine should be given to all women admitted with persistent vomiting.

Rehydration should be achieved with normal saline, supplemented with potassium chloride to address electrolyte imbalance. Care should be taken with dextrose-based fluids, as Wernicke's encephalopathy may be precipitated if there is thiamine deficiency.[44] Hyponatremia should be corrected slowly using normal saline to avoid central pontine myelinolysis. If a woman has symptoms of gastroesophageal reflux, proton pump inhibitors may provide additional benefit. Very rarely enteral feeding may be required if all other measures have failed, although a recent trial comparing enteral feeding with standard care did not demonstrate improvements in birth weight or maternal physical and psychological well-being.[45] Health care providers should be aware of

the high rates of suicidal ideation and consider referral for mental health support.[33]

Intrahepatic cholestasis of pregnancy

Introduction

ICP is the most common pregnancy specific liver disease, with reversible cholestasis with itching in pregnancy and raised fasting or post-prandial serum bile salts, with postpartum (within 6 weeks) relief of signs and symptoms. It has higher recurrence rate in following pregnancies. ICP has a variable rate among different regions, ranging from 3–5% of pregnant females in Chile, to 0.7% in the United Kingdom; its rare incidence in African region. ICP characteristically presents in the 3rd trimester of pregnancy but presentation has been seen as early as 7th week of pregnancy. Its incidence is more common in cases with multiple pregnancies and in females who received infertility treatment.

Etiology

The cause of ICP is not fully understood and is believed to be complex and influenced by various factors. Genetic predisposition, hormonal changes, and environmental factors are among the mechanisms proposed. Genetic susceptibility is supported by observations of the disease clustering in families, higher recurrence risk in subsequent pregnancies [46,47] and evidence of mutations in genes such as ABCB4, which encode multidrug resistance 3 (MDR3) proteins. These mutations often follow a sex-limited dominant pattern [48,49] of inheritance. Other genes implicated in ICP development include ATP8B1 (FIC1), ABCB11 (BSEP), ABCC2, and NR1H4 (FXR).[50-53]

Several studies have linked high estrogen levels to ICP, particularly in conditions like multiple pregnancies, ovarian hyperstimulation, contraceptive pill use, and late second-trimester presentation.[54-55] Additionally, low levels of selenium and vitamin D have been

associated with increased ICP prevalence, particularly in regions or seasons where these nutrients are scarce. [56-59]

While ICP is more common in women with underlying chronic liver diseases, it's uncertain whether these conditions contribute to ICP development or are simply unmasked by pregnancy.[60]

Clinical features

The primary complaint related to ICP is intense itching, typically beginning at the 30thweek of gestation. This itching is often more pronounced on the palmar and plantar surfaces with nocturnal worsening. Other symptoms may also be present, including nausea, loss of appetite, fatigue, discomfort in the upper right abdomen, pale stools and dark coloured urine. While clinical signs of jaundice are uncommon, it may develop in 14 to 25% of the patients within a month, once itching starts.[61] Some patients may also experience insomnia due to itching. Generally, examination findings are unremarkable other than the dermal scratch marks caused by itching. In the females who conceived through in-vitro fertilization, symptoms may appear transiently in the 1st trimester due to ovarian hyperstimulation, as compared to the persistent symptoms observed in the 2nd and 3rd trimesters in ICP.[55] It's important to consider a family history of liver disorders during pregnancy in other female relatives because of the role of genetic component involved in the pathophysiology of ICP.

Diagnosis

The diagnosis of ICP hinges on clinical symptoms, particularly itching in the 3rdtrimester, in conjunction with raised total serum bile salts, while ruling out other conditions that can present with similar symptomatic and laboratory abnormalities. If LFTs are elevated without pruritus, investigations for alternative causes are necessary. The most reliable marker for diagnosing ICP is total serum bile acids, with a sensitivity and specificity of 91 and 93 percent respectively when using a cut off value of 10 micromol/L.[62] Many studies utilize an upper limit of bile acids between 10 and 14 micromoles/L for diagnosing ICP.

Severe cholestasis, characterized by serum bile acid levels typically exceeding 40 micromol/L, increases the risk of fetal complications. Liver function tests such as ALT and AST are typically mildly elevated and usually do not surpass two times the upper limit of normal during pregnancy. Serum alkaline phosphatase levels may be elevated, possibly up to four times the upper normal value, but have limited diagnostic significance in ICP. Elevated bilirubin is present in approximately 25% of Study groups but rarely exceeds 6 mg/dL.[63] High prothrombin time may occur due to vitamin K deficiency, although postpartum hemorrhage is uncommon.[64]

Management

Ursodeoxycholic acid (UDCA), the main treatment for ICP, has a number of functional advantages that can enhance both the quality of life for mothers and fetuses. In addition to its anti-apoptotic qualities and improved biliary transit of bile acids, UDCA is thought to facilitate the elimination of pruritogens such as progesterone sulfates. According to research studies, UDCA lessens placental damage and enhances the transplacental transfer of bile acids from the fetus to the mother. Moreover, synergistic benefits have been demonstrated when UDCA is used with rifampicin, a strong pregnane X receptor agonist, particularly in situations when UDCA alone is unsuccessful. According to certain studies, S-adenosyl methionine can help alleviate pruritus and metabolic abnormalities.Nevertheless, dexamethasone should only be administered under obstetrician supervision to increase fetal lung maturity as it has not been shown to have any effect on symptoms or biochemical indicators in ICP. Cholestyramine may provide little symptomatic relief, but because it may reduce the absorption of vitamin K, potentially worsening perinatal outcomes, it should be used cautiously. Menthol-infused aqueous cream has been shown to benefit pruritus, whereas vitamin K supplementation may lower the risk of postpartum or neonatal hemorrhage.

ICP frequently reoccurs in future pregnancies and is linked to a heightened risk of hepatobiliary diseases later in life, including gallstones, hepatobiliary cancers, and immune-mediated and cardiovascular conditions. There is also a higher incidence of hepatitis C infection among women with ICP, though the cause-and-effect relationship is unclear. If symptoms or liver impairment persist beyond three months postpartum, further evaluation for other possible diagnoses is recommended.

Acute Fatty Liver of pregnancy

Introduction

AFLP, initially termed as ‘acute yellow atrophy of the liver’ in 1934, and continuously has been a significant obstetric emergency associated with notable maternal and perinatal mortality. It represents a severe condition marked by microvesicular fat deposition in the liver cells, typically occurring during the late stages of pregnancy, usually in the second or third trimester[65]. Symptoms commonly include vomiting, abdominal pain, and jaundice, often accompanied by polydipsia and polyuria. Although more prevalent in first-time mothers, AFLP can also affect women with previous uncomplicated pregnancies.

The clinical presentation of AFLP can vary widely. Patients may experience severe illness with manifestations such as encephalopathy, hypoglycemia, and/or ascites. LFTs typically reveal a mild to moderate elevation in aminotransferases along with jaundice. Often observed with coagulopathy, hyperuricemia, renal failure, and leucocytosis. While ultrasound scans may indicate fatty liver infiltration, this finding lacks sensitivity and specificity. The definitive diagnosis often relies on liver biopsy, which demonstrates diffuse or peri-venular microvesicular steatosis.

Recent AFLP related studies have cast light on its incidence. A recent study conducted across 229 hospitals in the UK, covering 1,132,964 pregnancies, estimated an incidence of 5 cases for every 100,000 pregnancies[66]. Another UK-based hospital study reported 5 cases of AFLP for every 4377 pregnancies[1]. A prospective study from a tertiary-care hospital in our country indicated an incidence of 30 AFLP cases for every 100,000 pregnant females.[67]

Pathogenesis [68,69]

AFLP represents a mitochondrial hepatopathy akin to conditions like Reye-Johnson syndrome and drug-induced toxicity such as from valproic acid. It stems from a defective beta-oxidation of fats in mitochondrial. The mother typically harbours a compensated defective utilization of fatty acid, which becomes apparent in later months of pregnancy when reliance on fatty acid metabolism for energy increases. There's been a suggestion of an association between maternal hepatic disorder and fetal FAO related disorders. As late pregnancy places greater reliance on fats being chief energy source, it creates a scenario where liver disease phenotypically manifests overtly, even in previously asymptomatic and healthy individuals.

AFLP and rest of the liver disorders associated to pregnancy are more prevalent when the fetus carries composite heterozygous or a homozygous defect in enzymes which carry out fatty acid oxidation (FAO)[70,71]. These defects, which are autosomal recessive, commonly involve mutations in the LCHAD (long-chain hydroxyacyl coA dehydrogenase) part of mitochondrial tri-functional protein (MTP). While mutations in other FAO pathway enzymes, such as carnitine palmitoyl transferase[72], have also been sporadically reported in AFLP patients, LCHAD mutations are most frequently observed.

The FAO enzyme defect in the fetus substantially increases maternal susceptibility to AFLP and disorders related to pregnancy[73]. However, this is not a law, as not all pregnant females with AFLP carry these

known mutations[74,75] and all mothers carrying fetuses with homozygous enzymatic defects do not develop AFLP[73]. This suggests a heterogeneous pathogenetic mechanism for AFLP.

Considering that the placenta has the same genotype as the fetus and that most pregnant females with AFLP experience rapid recovery after delivering the fetus/placenta, researchers have investigated how the placenta is playing a key role in the pathogenesis of AFLP.

A defective FAO within the placental mitochondria of pregnant femaleswith AFLP results in dysfunctional mitochondrialeading to elevated placental and serum oxidative/nitrosative stress. Simultaneously, the faulty FAO leads to the buildup of toxic intermediates, such as free fatty acids like arachidonic acid, in the serum of these patients and the placenta. These mechanisms have been demonstratedby conducting a number of experiments in our laboratories. Furthermore, our research has shown that incubating liver cells with concentration of arachidonic acid equivalent as found in the serum of AFLP patients is sufficient to induce mitochondrial damage, apoptosis, and lipid accumulation in vitro.[69]

Clinical Features

Table 4: Swansea Criteria for the Diagnosis of Acute Fatty Liver of Pregnancy[24,26]

Six of the features below are required for the diagnosis	
Clinical features	▪ Nausea and vomiting ▪ Abdominal pain ▪ Encephalopathy ▪ Polyuria or polydipsia
Laboratory features	▪ Bilirubin >0.8 mg/dL ▪ Hypoglycemia <72 mg/dL ▪ WBC >11x10^9/L ▪ AST or ALT >42 units/L ▪ AKI or Cr >1.7 mg/dL ▪ Coagulopathy or PT >14 sec ▪ Ammonia >47 μmol/L ▪ Urate >340 μmol/L
Ultrasonographic features	Ascites or echogenic liver
Histologic features	Microvesicular steatosis on liver biopsy

AKI, acute kidney injury; ALT, alanine transaminase; AST, aspartate transaminase; Cr, creatinine; PT, prothrombin time; WBC, white blood cell count.

Management

Once diagnosed with acute fatty liver of pregnancy (AFLP), delivery of the fetus, regardless of gestational age, is the sole treatment. Delivery invariably leads to the resolution of hepatic dysfunction and associated complications.

The mode of delivery depends on several factors:

- Fetal status: Close monitoring is essential as many fetuses show signs of asphyxia and hypoxia. Prompt delivery may be necessary if fetal compromise is evident [5].
- Maternal coagulation status: Coagulation abnormalities may necessitate replacement of coagulation factors if cesarean delivery is required.
- Likelihood of successful labor induction: If delivery cannot be achieved safely within 24 hours of diagnosis, cesarean delivery may be preferable.
-

Management of severe hypoglycemia is crucial to prevent coma and death. Patients require a 5% dextrose solution to maintain blood glucose levels, with close monitoring until hepatic function improves and the patient can tolerate a regular diet.Renal function may also be affected, potentially leading to acute tubular necrosis and hepatorenal syndrome due to factors such as maternal hemorrhage. Close monitoring of fluid balance is essential to prevent pulmonary edema resulting from low plasma oncotic pressures.

In severe Study groups, postpartum plasma exchange has been utilized, particularly for patients with severe encephalopathy, ventilator support, or liver/renal insufficiency unresponsive to conventional management. Studies have shown improvement in signs and laboratory values with plasma exchanges a standalone option or along with continuous hemodiafiltration, for patients with multiple organ dysfunction resulting from AFLP during pregnancy.

Preeclampsia/HELLP

Hypertensive disorders of pregnancy represent a significant global cause of maternal and perinatal mortality, with preeclampsia/eclampsia complicating 2% to 8% of pregnancies and contributing to 25% of serious maternal complications [76, 77, 78]. Pre-eclampsia can be defined as a condition characterized by new-onset hypertension,>20thweek of pregnancy (blood pressure ≥140/90 mmHg) accompanied by proteinuria (>300 mg/day) and organ dysfunction such as hepatorenal insufficiency, neurological or hematological complications. The occurrence of seizures distinguishes pre-eclampsia from eclampsia. HELLP syndrome is regarded as a severe manifestation of pre-eclampsia.

The occurrence of preeclampsia in the USA has increased by 25% between 1987 and 2004[79], and severe cases number has rose nearly sevenfold during this time[80] This rise in preeclampsia cases may be associated with the growing prevalence of risk factors like older maternal age, obesity, diabetes, and pre-existing high blood pressure.

HELLP syndrome was initially identified in 1982 as a syndrome characterized by thrombocytopenia, elevated liver enzymes, and hemolysis. Incomplete HELLP syndrome occurs when only one or two of these symptoms are present .Approximately 10% to 20% of individuals with preeclampsia develop HELLP syndrome, which can manifest even with no sign of hypertension or proteinuria[76,81].In a study conducted in Switzerland involving 241 cases of preeclampsia, 17% also had HELLP syndrome, whereas only 3% experienced isolated HELLP syndrome[81]. Similarly, a French study involving 435 cases of preeclampsia, 28% had HELLP syndrome, with only 4.4% presenting with isolated HELLP syndrome.[82] Notably, a quarter of HELLP syndrome cases occur postpartum[83], typically within 48 hours following childbirth[84].

Risk factors for preeclampsia encompass nulliparity, multifetal gestations, previous occurrences of preeclampsia, a positive family history, advanced maternal age, and pregnancies conceived through assisted reproductive technology. Additionally, certain maternal comorbidities, such as pre-existing kidney or vascular conditions, autoimmune diseases like lupus, chronic hypertension, pregestational or gestational diabetes, obesity, intrahepatic cholestasis of pregnancy (ICP), and antiphospholipid antibody syndrome, heighten the risk. HELLP syndrome shares similar risk factors with preeclampsia, with older mothers (above 35 years) being at the highest risk[85,86].

The placenta plays a pivotal role in the pathophysiology of preeclampsia, as the condition typically resolves upon delivery of the placenta. Preeclampsia is fundamentally a disorder related to abnormal placentation. It is hypothesized that inadequate invasion of the trophoblast into the uterine lining leads to impaired arterial placental perfusion. This impairment worsens as the pregnancy progresses and the demands on the placenta increase.

Numerous chemicals, including prostaglandins, endothelin, and nitric oxide, are released by placental tissue and are linked to endothelial dysfunction, arterial hypertension, and platelet aggregation. Microangiopathic haemolytic anemia is caused by the creation of cross linked networks in small blood vessels as a result of endothelial injury causing the release of fibrin. One possible explanation for liver involvement in preeclampsia is secondary fibrin deposition in the hepatic sinusoids, which can clog the sinuses and lead to hepatic ischemia. Hepatic rupture, parenchymal bleeding, and subcapsular hematoma development might result from the combination of ischemia and hepatic sinusoidal obstruction.

Table 5- The Tennessee and Mississippi classification of HELLP is shown in supplementary [12]

HELLP Class	Mississippi Classification	Tennessee Classification
1	Platelet count ≤50,000/μL AST or ALT ≥70 IU/L LDH ≥ 600 IU/L	Platelet count ≤100,000/uL AST ≥ 70 IU/L LDH ≥ 600 IU/
2	50,000/μL ≤Platelet count ≤100,000/μL AST or ALT ≥70 IU/L LDH ≥ 600 IU/L	N/A
3	100,000/μL ≤ Platelet count ≤ 150,000/μL AST or ALT ≥40 IU/L LDH ≥ 600 IU/L	
Partial HELLP/ incomplete HELLP	N/A	(Severe preeclampsia + one of the following: ELLP, EL, LP)*

Clinical features

Severe headache, blurred vision or scotoma, epigastric or right upper quadrant pain, nausea and vomiting, and sudden swelling of the face, hands, or feet are the most commonly reported symptoms of HELLP syndrome. The presentation of HELLP can be atypical; up to 19% of patients may be normotensive[76,81], and absence of proteinuria in 15% cases. While overt DIC is rare in preeclampsia or eclampsia alone, it has incidence of 6% to 32% among females with HELLP syndrome.[84,87,88]

Abdominal ultrasound to assess the liver size, hepatic vasculature patency, and signs of portal hypertension, is indicated. Hepatic dysfunction in preeclampsia is marked by elevated aminotransferase levels, with AST typically higher than ALT initially, possibly due to the centrifugal nature of liver injury and contributions from hemolysis in HELLP Study groups. Median ALT levels are around 200 U/L[89,90,91,92], although severe preeclampsia/HELLP syndrome can cause much

higher levels (1000–5000 U/L), particularly if hepatic infarction occurs[93]. Other notable features include increased lactate dehydrogenase (from liver and red blood cells), elevated uric acid, and indirect hyperbilirubinemia due to hemolysis. Severe cases may also present with increased prothrombin time and evidence of DIC. Ascites, which can be detected through imaging or clinically, are often transient and typically present with fluid characteristics consistent with portal hypertension.[94,95,96]

Liver dysfunction is generally less severe in preeclampsia/HELLP syndrome compared to AFLP. In a study conducted between 2004 and 2020 involving 101 women, 21 with AFLP and 80 with HELLP syndrome, significant differences in maternal complications were observed. Jaundice occurred in 86% of AFLP cases, compared to 14% of HELLP cases. Acute kidney injury was seen in 62% of AFLP cases versus 15% in HELLP cases. Disseminated intravascular coagulation (DIC) was present in 67% of AFLP cases compared to 9% in HELLP cases, and sepsis occurred in 48% of AFLP cases versus 10% in HELLP cases.[97]

Maternal and Fetal Outcomes

Maternal mortality associated with preeclampsia was 6 per 100,000 pregnancies in a contemporary UK cohort. [98] Maternal risks are correlated with the severity of preeclampsia, with higher risks observed in those with HELLP syndrome compared to those without extra-placental involvement. Maternal mortality rates for HELLP syndrome are reported to be between 2% and 10%, and perinatal mortality ranges from 0% to 15%, with higher rates noted in older studies from less economically affluent countries. [99,100] Complications related to HELLP syndrome account for approximately 80% of the mortality in women with preeclampsia[100]. Maternal and perinatal mortality rates are 10 and 4.5 times higher, respectively, in women with HELLP syndrome compared to those without. The incidence of acute liver failure is 4.5% in HELLP patients versus 0.1% in non-HELLP controls

[101]. Direct complications of HELLP syndrome include abruptio placenta (9%–20%), DIC (5%–56%), and acute renal failure (7%–36%)[101], with DIC being significantly higher in cases involving abruptio placenta[87].Less frequent complications of HELLP syndrome include eclampsia (4%–9%), pulmonary edema (3%–10%), and subcapsular liver hematoma (<2%)[101]. Hepatic rupture, though rare, occurs in 0.53% to 2% of women with preeclampsia and/or HELLP syndrome[102,103]. The right lobe of the liver is most commonly affected, in 75% or more cases[104]. Recent reviews report a maternal mortality rate of 7.5%, down from previous estimates of 17% and 32%[105], attributed to improved management practices. Late diagnosis is considered the primary contributor to poor outcomes, with perinatal mortality around 40%.[103]

Treatment

The sole proven treatment for pre-eclampsia is placenta delivery, which is the mainstay of supportive care. Consequently, the fetus ought to be delivered as soon as possible, particularly in situations when the pregnancy is past 34 weeks gestation, there is recorded fetal distress, or there is indication of maternal decline. Intravenous labetalol, intravenous hydralazine, and oral nifedipine are the first-line treatments for hypertension in pregnant women. To encourage fetal lung maturity, glucocorticoids should be given if the gestational age is less than 34 weeks. In certain situations, coagulation profiling and assistance may be necessary. The initial line of treatment for severe pre-eclampsia with seizures and HELLP syndrome is magnesium sulfate, or MgSO4.Heart disease and stroke are twice as likely to occur in later life in women with pre-eclampsia and eclampsia, though it's unclear if this is solely a connection, causative, or a combination of the two. Women with HELLP syndrome most likely face comparable risks, albeit this hasn't been proven beyond a reasonable doubt.

Pre-existing liver diseases and pregnancy

Due to metabolic and endocrine problems, cirrhosis and portal hypertension have a substantial negative influence on women's fertility, making pregnancy difficult. Non-ovulation, amenorrhea, and infertility are caused by interference with the hypothalamic-pituitary axis and changes in the metabolism of estrogen. Preterm labor, perinatal mortality, and spontaneous fetal loss are all higher in pregnant women with cirrhosis. Pregnancy may worsen liver disease in the mother, increasing the risk of encephalopathy, ascites and variceal bleeding, In the early 1980s, maternal mortality for cirrhosis-affected pregnant women reached as high as 10.5%; however, in more recent times, this rate dropped to 1.6%, with a 10% decompensation rate

Pregnancy outcomes are primarily determined by the degree of the mother's liver condition rather than its underlying cause. Predicting the likelihood of maternal decompensation during pregnancy and directing preconception counselling for women with cirrhosis are two important uses of prognostic scoring systems, such as the MELD (Model for End-stage Liver Disease) and UKELD (United Kingdom Model for End-stage Liver Disease). In a study done in 62 pregnant females, 29 women with cirrhosis had a 10% hepatic decompensation rate.

When a pregnant patient with cirrhosis has portal hypertension, the primary cause of maternal death is variceal hemorrhage. Due to increasing blood volume and the gravid uterus pressing on the inferior vena cava, which reduces venous return, portal hypertension develops throughout pregnancy and peaks in the second trimester. Pregnant women who have a history of varices are at a 25% chance of experiencing variceal hemorrhage, which can be fatal and occur most often during the second trimester and birth.

Pregnancy-related portal hypertension management is difficult, with death rates as high as 50%. There are no set standards for variceal hemorrhage management, prevention, or screening. For "at-risk"

varices prior to conception, prophylactic endoscopic variceal banding may be explored, albeit this is not conclusively proven. Beta-blockers should be used for variations lacking high-risk characteristics because the advantages usually outweigh the hazards. Since upper gastrointestinal endoscopy is generally safe during pregnancy, the American Association for the Study of Liver Diseases recommends that women with cirrhosis receive endoscopic screening during the second trimester. Treatment for identified varices should adhere to established protocols. According to new guidelines, patients with cirrhosis who might benefit from endoscopic screening for varices should be identified using non-invasive indicators. According to recent data, a platelet count of less than 110,000 can be use as an indicator whether pregnant women needs endoscopic screening or not.

Mishra et al.[16] conducted a study to evaluate the impact of maternal liver disorders on fetal outcomes. They performed thorough clinical assessments, including detailed histories and examinations of the patients. The study assessed 1,000 pregnancies, finding that 200 pregnant women had liver diseases, leading to 20% prevalence. Among these, Pre-eclampsia/HELLP were the most common, affecting 142 women (62%), followed by intrahepatic cholestasis, which affected 34 women (17%).

The fetal outcomes observed included a prematurity rate of 28.5%, a stillbirth rate of 14.5%, and early neonatal death occurring in 11% of the cases. Additionally, 21% of the newborns required admission to the neonatal intensive care unit (NICU). The newborns were premature in most number of patients.

S. P. Pereira et al.[106] conducted a study on liver disorders in pregnancy, focusing on AFLP and HELLP syndrome.Over a span of 10 years, the study observed 46 women with hepatic dysfunction severe enough to necessitate transfer to a Liver Failure Unit. The median age of the women was 30 years, ranging from 17 to 41 years. Three-

quarters of the women were nulliparous, and five had twin pregnancies. The median gestational age at admission was 35 weeks, ranging from 24 to 40 weeks.

Upon admission, 32 patients (70%) were diagnosed with preeclampsia, and 21 (46%) showed signs of encephalopathy and/or required ventilation. Among them, 32 patients (70%) exhibited clinical features and laboratory values consistent with AFLP, while 7 (15%) had HELLP syndrome. One patient experienced liver rupture due to preeclampsia, necessitating liver transplantation. In six other patients, severe liver dysfunction not related to pregnancy was identified. Infectious complications were observed in 17 patients with AFLP (53%) and 2 with HELLP syndrome (29%). Major intra-abdominal bleeding occurred in 12 women, 10 of whom had AFLP, with 9 requiring laparotomies for clot evacuation.

Four patients with AFLP (12.5%) had fatal outcomes, causing a perinatal mortality rate of 9%. However, no maternal or perinatal deaths were linked with HELLP syndrome. Contrary to findings from previous studies, this large series suggests a relatively favourable maternal and perinatal outcome in severe cases of AFLP and HELLP syndrome. The study suggests that further improvements in outcomes are likely achievable through the prevention of bleeding and infectious complications associated with these disorders.

Garcia Romero et al.[107] reported findings from a tertiary referral women's hospital, 11.24% females presented with hepatic disorders out of total pregnant females came to the hospital, contrasting with the global prevalence of 3%. Associated liver disease was identified in 10.8% of total pregnant females,Pre-eclampsia was associated with 9.9% of pregnant females. Only 0.56% were attributed to hepatic disorder that was coincidental or pre-existing, with acute or chronic hepatitis C virus being the commonest in this subgroup (0.12%).

The study spanned three years, during which 10,762 pregnant females came to hospital, out of which 11.33% had hepatic disorders. This high number may be due to the hospital's specialization in managing high-risk pregnancies. Among the cases, 10.81% were identified with pregnancy-related liver diseases, including1.12% patients with pre-existing hepatic disorder (13 cases).

Pre-eclampsia emerged as the commonest affecting 1070 pregnant women, occurring as an isolated cases and in association with other hepatic pathologies in 5 patients. Thirty four cases of HELLP syndrome (0.32%), while 40 cases had ICP (0.37%), and 6 cases presented with AFLP (0.06%).

The frequency of ICP in their sample was lower than reported in Chile which is similar to Latin group living in the US. No cases of HG were recorded during the study period, except for one patient with tumor in liver. Hepatopathies coinciding with pregnancy or pre-existing conditions were seen in 60 cases (0.56%), either as standalone diseases in 47 cases (0.44%) or in conjunction with pregnancy-related liver diseases in 13 patients (0.12%). Commonest causes found to be Viral hepatitis, particularly hepatitis C virus (HCV), and non-alcoholic fatty liver disease (NAFLD) in this group.

MATERIAL AND METHODS

- A prospective study was conducted in 100 pregnant females with liver disease and 50 controls (pregnant females without liver disease) admitted in department of Medicine and Gynaecology in a tertiary care hospital,Dayanand Medical College and Hospital,Ludhiana during a 15 month period from 15th september,2022 to 15th December,2023.
- Liver function tests were studied and compared between the two groups.
- Total number of pregnancies was noted during the 15 month period, out of which females fulfilling the criteria were considered for the study.

SOURCE OF DATA

Pregnant females with liver disease admitted at Medicine and Gynaecology wards at Dayanand medical college and hospital, Ludhiana

METHOD OF COLLECTION OF DATA

- **INCLUSION CRITERION (FOR STUDY GROUP**) –

  1) Female patients older than 21 years of age,willing and able to sign consent
  2) UPT +ve or USG Evidence of ongoing Pregnancy
  3) Liver enzymes > 2 times elevated

- **EXCLUSION CRITERION –**

  1) Non pregnant females
  2) Age < 21 years
  3) Not able to give consent
  4) Liver enzymes < 2 times elevated
  5) Any other chronic disease- Chronic kidney disease/Chronic obstructive airway disease/Coronary artery disease,Epilepsy

- **INCLUSION CRITERION (FOR CONTROL GROUP)** - Pregnant females > 21 years of age with < 2 times elevated liver enzymes

- **Duration of Study** - 15-09-2022 TO 15-12-2023

- **Number of Study group** – 100

- **Number of control group** – 50

CRITERIA FOR DIAGNOSING INTRAHEPATIC CHOLESTATSIS OF PREGNANCY [108]

Positive criteria

1. Itching during pregnancy.
2. Disappearance of itching after delivery.
3. Itching during previous pregnancy or after contraceptive pills in patients, mother or sister.
4. Itching should be associated with deranged LFT. Sometimes liver function test becomes deranged after 1 or 2 weeks of itching.

Negative criteria

1. No signs of dermatological disease.
2. No recurrent itching except during pregnancy or use of contraceptive pills 3. No other active liver disease.

CRITERIA FOR DIAGNOSING HYPEREMESIS GRAVIDARUM [109]

- Nausea and vomiting of unusual severity requiring admission upto 20 weeks period of gestation along with weight loss exceeding 5% of pre pregnancy body weight and ketonuria unrelated to any other cause.
- All other causes of nausea and vomiting ruled out.

CRITERIA FOR DIAGNOSIS OF VIRAL HEPATITIS [108]

1) Evaluation in terms of history, physical examination and course of disease.
2) Recent onset of jaundice with conjugated hyperbilirubinemia.
3) Raised aminotransferases
4) ELISA marker for viral hepatitis
5) Exclusion of other diseases like exposure to drugs,toxins known to induce liver disease, cholestatic jaundice of pregnancy and eclampsia.

CRITERIA FOR DIAGNOSIS OF HELLP SYNDROME [109]

1. Hemolysis :-any 2 of the following:
   a. Serum bilirubin >1.2mg/dl
   b. Peripheral blood film showing schistocytes or helmet cells
   c. Increased LDH (>600 mg/dl) or decreased haptoglobin (<25mg/dl)
   d. Presence of severe anemia not explained by any other cause
2. Elevated Liver enzymes SGOT/PT >70 IU
3. Platelet count < 1 lakh /mm3

CRITERIA FOR THE DIAGNOSIS OF ACUTE FATTY LIVER OF PREGNANCY [108]

1. Increased liver enzymes 5 to 10 times the upper limit
2. Increased serum bilirubin.
3. Increased serum creatinine.
4. Increased white cell count
5. Increased ammonia
6. Deranged coagulation profile.
7. Decreased glucose levels.
8. Peripheral blood film show fragmented red cells, burr cells, helmet cells or schistocytes

Outcome: The results will be evaluated both in terms of maternal and fetal outcomes as-

1. MATERNAL

a) Normal outcome

b) Prolonged Hospital Stay (defined as > 2 Days In Normal Vaginal Delivery and > 4 Days In Cesarean Section)

c) Multiorgan Failure

d) Maternal Mortality

2. FETAL

1. Normal Live Birth
2. Preterm Delivery
3. Intrauterine Death
4. Abortion
5. Fetal Growth Restriction

3. STATISTICAL ANALYSIS -Data will be described in terms of range; mean ± standard deviation (± SD), median, frequencies (number of Study groups) and relative frequencies (percentages) as appropriate. To determine whether the data is normally distributed, a Kolmogorov-Smirnov test will be used. Comparison of quantitative variables between the study groups will be done using Student t-test and Mann Whitney U test for independent samples for parametric and non-parametric data respectively. For comparing categorical data, Chi square ($\chi 2$) test will be performed and fisher exact test will be used when the expected frequency is less than 5. A probability value (p value) less than 0.05 will be considered statistically significant. All statistical calculations will be done using (Statistical Package for the Social Science) SPSS 21version (SPSS Inc., Chicago, IL, USA) statistical program for Microsoft Windows.

OBSERVATIONS AND RESULTS

EPIDEMIOLOGY

TABLE 6- DISTRIBUTION OF STUDY-GROUP AND CONTROL-GROUP ACCORDING TO AGE

		STUDY GROUP		CONTROL GROUP		Total	Chi-square value	p-value
AGE GROUP		STUDY GROUP	STUDY GROUP	CONTROL GROUP	CONTROL GROUP			
	< 25	18	18.0%	7	14.0%	25	4.844	0.184
	26-30	36	36.0%	20	40.0%	56		
	31-35	24	24.0%	18	36.0%	42		
	> 35	22	22.0%	5	10.0%	27		
Total		100	100.0%	50	100.0%	150		

In our study, out of 100 patients in study-group,18 patients were in <25 years age group (18.0 %),36 patients in 26-30 years age group (36.0%),24 patients in 31-35 years age group (24.0%) and 5 patients in > 35 years age group (22.0 %) while In control group , 7 patients were in <25 years age group (14.0 %),20 Patients in 26-30 years age group (40.0 %),18 patients in 31-35 years age group (42.0%) and 5 patients in > 35 age group (10.0%) with p value of 0.184 as shown in Table 6 ,Figure 1

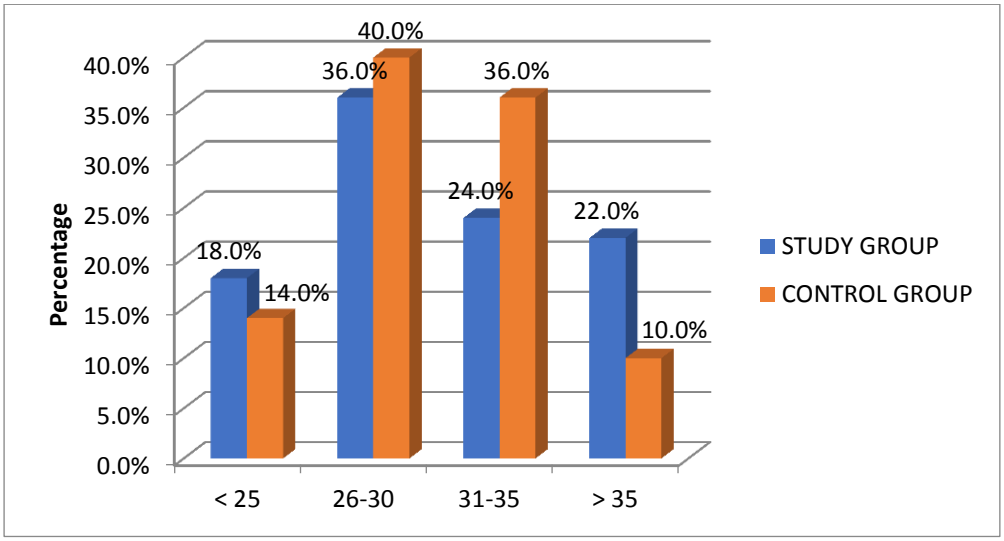

FIGURE 1- DISTRIBUTION OF STUDY-GROUP AND CONTROL GROUP ACCORDING TO AGE

TABLE 7- DISTRIBUTION OF STUDY-GROUP ACCORDING TO ETIOLOGY

STUDY-GROUP	No.	%age
Mild ICP	20	20.0%
Moderate ICP	17	17.0%
Tropical Fever	11	11.0%
Severe ICP	8	8.0%
HELLP	6	6.0%
Pre-eclampsia	5	5.0%
Acute Viral Hepatitis	5	5.0%
Acute Fatty liver of Pregnancy	3	3.0%
Hyperemesis Gravidarum	3	3.0%
Acute Liver Failure	2	2.0%
Acute on Chronic Liver Failure	2	2.0%
Drug Induced Liver Injury	2	2.0%

In our study, Out of 100 patients,45 patients had ICP, out of which 20 patients had Mild ICP (20.0%),17 patients had Moderate ICP (17.0%),8 patients presented with Severe ICP (8.0%),11 patients had Tropical Fever (11.0%),6 patients had HELLP (6.0%),5 patients had Pre-eclampsia (5.0%),5 patients had Acute Viral Hepatitis (5.0%),3 patients had Acute fatty liver of pregnancy(3.0%),3 patients had Hyperemesis Gravidarum (3.0%),2 patients presented with Acute Liver Failure (2.0%), 2 patients had Acute on Chronic liver failure (2.0%) and 2 patients had Drug induced Liver Injury (2.0%) caused by progesterone as shown in Table 7,Figure 2

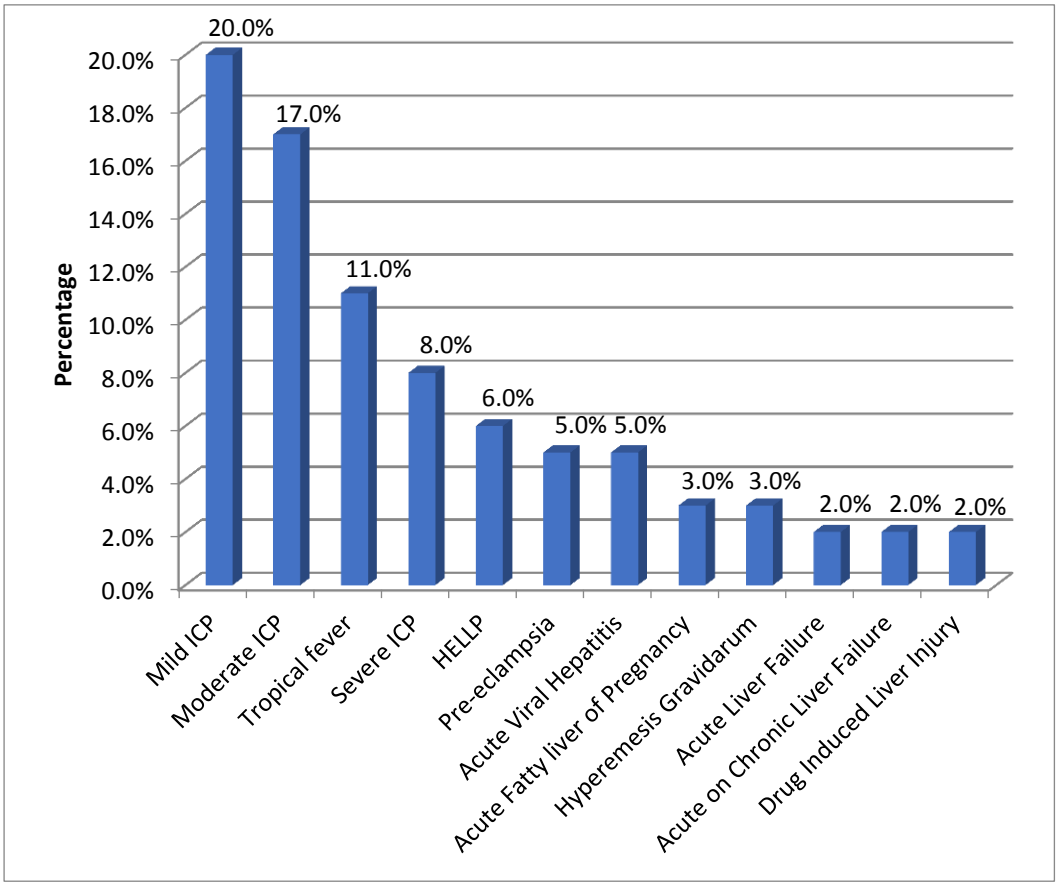

FIGURE 2 - DISTRIBUTION OF STUDY- GROUP ACCORDING TO ETIOLOGY

TABLE 8 -DISTRIBUTION OF STUDY-GROUP AND CONTROL GROUP ACCORDING TO TRIMESTER

		STUDY GROUP		CONTROL GROUP		Total	Chi-square value	p-value
		STUDY GROUP	STUDY GROUP	CONTROL GROUP	CONTROL GROUP			
Trimester	1st	4	4.0%	0	0.0%	4	9.021	0.011
	2nd	16	16.0%	1	2.0%	17		
	3rd	80	80.0%	49	98.0%	129		
Total		100	100.0%	50	100.0%	150		

In our study, In study group,out of 100 patients , 4 patients presented in 1st trimester (4.0%).16 patients presented in 2nd trimester (16.0%) and 80 patients presented in 3rd trimester (80.0%) While In control group, out of 50 patients, no patient presented in first trimester, 1 patient presented in 2nd trimester (2.0%) and 49 patients presented in 3rd trimester (98.0%).p-value was 0.011 as shown in Table 8,Figure 3

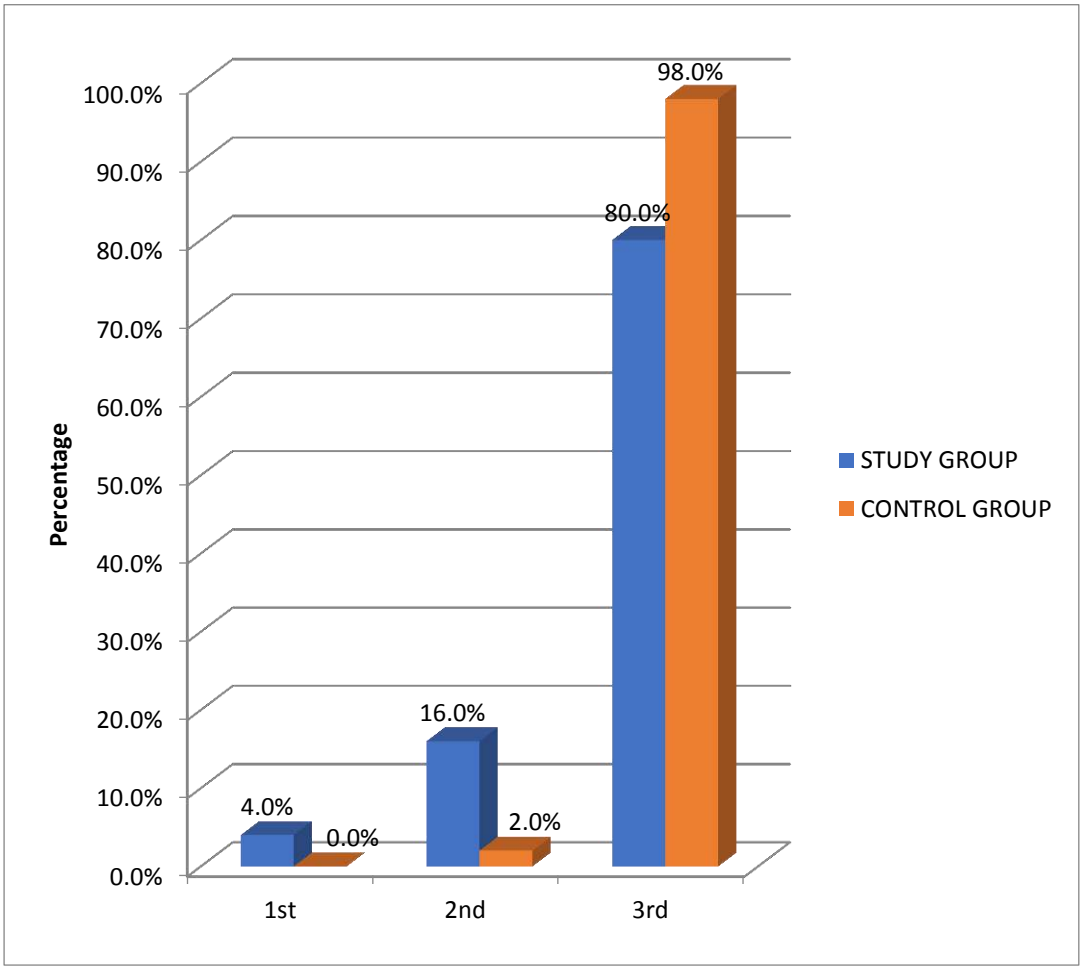

FIGURE 3 - DISTRIBUTION OF STUDY-GROUP AND CONTROL GROUP ACCORDING TO TRIMESTER

TABLE 9 - DISTRIBUTION OF STUDY-GROUP AND CONTROL GROUP ACCORDING TO GESTATIONAL AGE

		STUDY GROUP		CONTROL GROUP		Total	Chi-square value	p-value
		STUDY GROUP	STUDY GROUP	CONTROL GROUP	CONTROL GROUP			
Gestation Age (in weeks)	< 28	20	20.0%	1	2.0%	21	21.706	0.001
	28-34	20	20.0%	4	8.0%	24		
	34-36	15	15.0%	3	6.0%	18		
	> 36	45	45.0%	42	84.0%	87		
Total		100	100.0%	50	100.0%	150		

In our study,in study-group,Gestational age was < 28 weeks in 20 patients (20.0%), 28-34 weeks in 20 patients (20.0%) ,34-36 weeks in 15 patients (15.0%) and >36 weeks in 45 patients (45.0%).In control group, Gestational age was < 28 weeks in 1 patient (2.0%), 28-34 weeks in 4 patients (8.0%) ,34-36 weeks in 3 patients (6.0%) and >36 weeks in 42 patients (84.0%).p-value was 0.001 as shown in Table 9,Figure 4

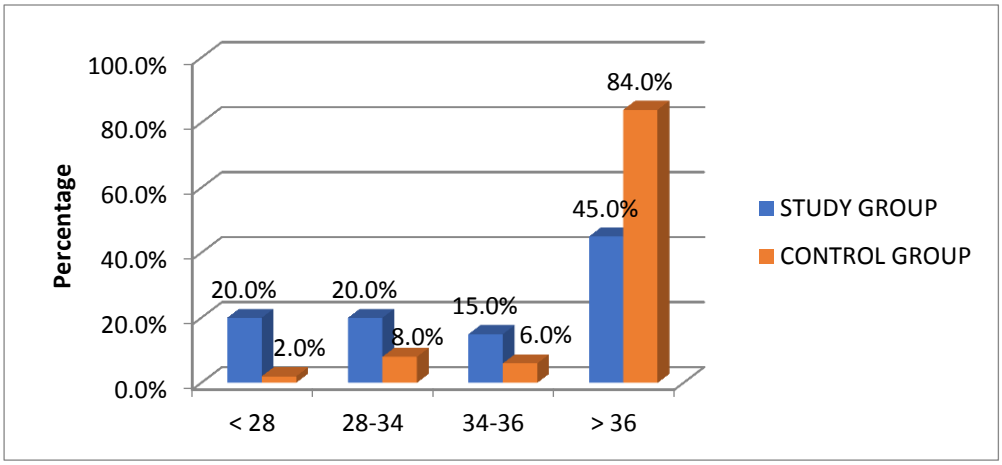

Figure 4- DISTRIBUTION OF STUDY-GROUP AND CONTROL GROUP ACCORDING TO GESTATIONAL AGE

TABLE 10 - MEAN GESTATIONALAGE IN OUR STUDY

	STUDY GROUP		CONTROL GROUP		Z	p-value
	Mean	SD	Mean	SD		
Gestation Age (in weeks)	32.14	7.11	36.78	2.95	-5.704	0.001

In our study, Mean Gestational age in study group was 32.17 weeks .In control group, Mean Gestational age was 36.78 weeks with p value of 0.001 as shown in Table 10 ,Figure 5

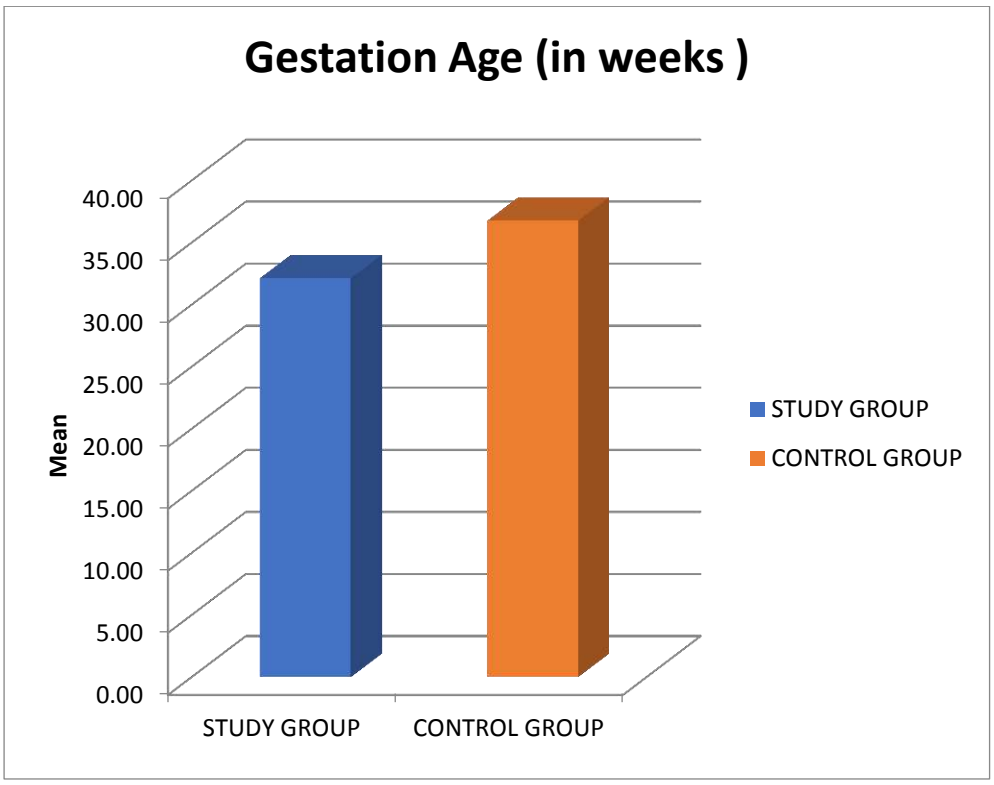

FIGURE 5 - MEAN GESTATIONAL AGE IN OUR STUDY

TABLE 11 - DISTRIBUTION OF STUDY-GROUP AND CONTROL GROUP ACCORDING TO COMORBIDITIES

		STUDY GROUP		CONTROL GROUP		Total	Chi-square value	p-value
		Study Group	Study Group	Control Group	Control Group			
Co Morbidities (DM/HTN/HYPOTHYRODISM)	**No**	81	81.0%	43	86.0%	124	0.582	0.446
	Yes	19	19.0%	7	14.0%	26		
Total		100	100.0%	50	100.0%	150		

In our study, Comorbidities were present in 19 patients (19.0%) and were absent in 81 patients (81.0%).In control group Comorbidities were present in 7 patients (14.0%) and were absent in 43 patients (86.0%).p-value was 0.446 as shown in Table 11,Figure 6

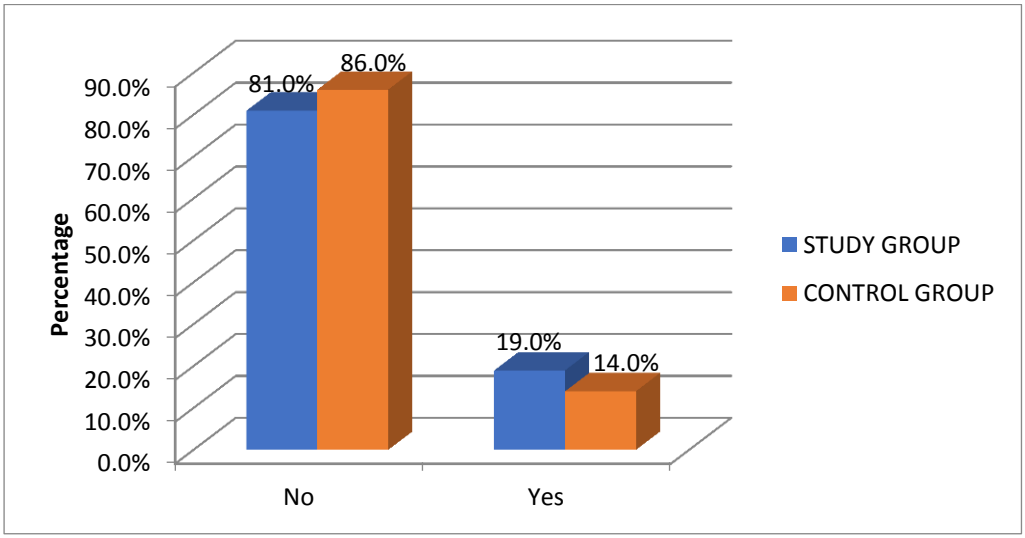

FIGURE 6-DISTRIBUTION OF STUDY-GROUP AND CONTROL GROUP ACCORDING TO COMORBIDITIES

TABLE 12- DISTRIBUTION OF STUDY-GROUP AND CONTROL GROUP ACCORDING TO GRAVIDA

		STUDY GROUP		CONTROL GROUP		Total	Chi-square value	p-value
		Study Group	Study Group	Control Group	Control Group			
Gravida	**Multi gravida**	59	59 %	35	70.0%	94	1.724	0.189
	Primi gravida	41	41 %	15	30.0%	56		
Total		100	100 %	50	100.0%	150		

In our study, In study-group 59 patients were multigravida (59.0 %) and 41 patients were primigravida (41.0%) While In control group, 35 patients were multigravida (70.0 %) and 15 patients were primigravida (30.0%).p-value was 0.189 as shown in Table 12 ,Figure 7

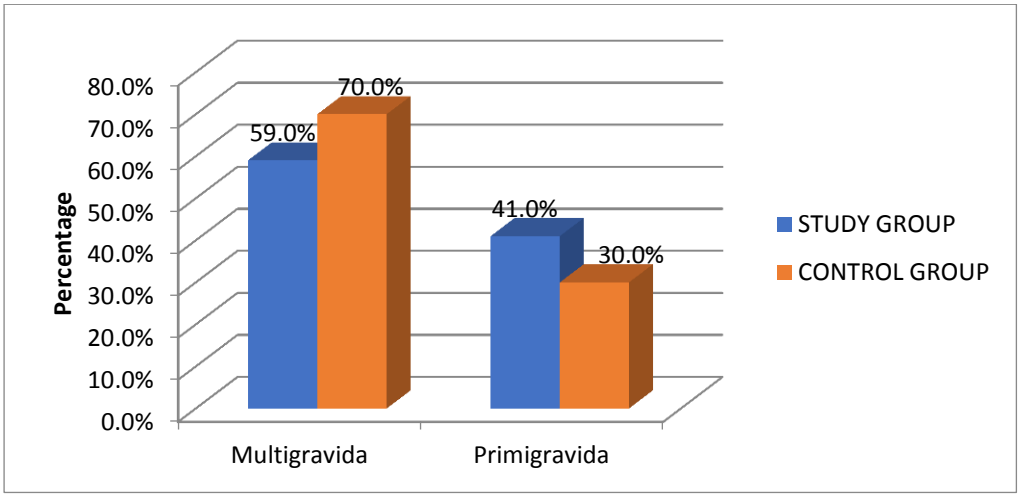

FIGURE 7 - DISTRIBUTION OF STUDY-GROUP AND CONTROL GROUP ACCORDING TO GRAVIDA

TABLE 13 - DISTRIBUTION OF STUDY-GROUP AND CONTROL GROUP ACCORDING TO SYMPTOMS

		STUDY GROUP		CONTROL GROUP		Total	Chi-square value	p-value
		No.	%age	No.	%age			
Fever	No	94	94.0%	50	100.0%	144	3.125	0.077
	Yes	6	6.0%	0	0.0%	6		
Persistent Nausea/Vomiting	No	92	92.0%	50	100.0%	142	4.225	0.052
	Yes	8	8.0%	0	0.0%	8		
Yellowish Discoloration	No	86	86.0%	50	100.0%	136	7.721	0.005
	Yes	14	14.0%	0	0.0%	14		
Pain Abdomen	No	51	51.0%	14	28.0%	65	7.181	0.007
	Yes	49	49.0%	36	72.0%	85		
Itching	No	88	88.0%	49	98.0%	137	4.211	0.061
	Yes	12	12.0%	1	2.0%	13		
Difficulty in Breathing	No	97	97.0%	50	100.0%	147	1.531	0.551
	Yes	3	3.0%	0	0.0%	3		
Decreased Fetal Movements	No	80	80.0%	44	88.0%	124	1.489	0.222
	Yes	20	20.0%	6	12.0%	26		
Bleeding Per Vaginum/Spotting	No	78	78.0%	47	94.0%	125	6.144	0.018
	Yes	22	22.0%	3	6.0%	25		

In our study,In Study group 6 patients presented with fever (6.0%),8 patients had nausea and vomiting (8.0%),14 patients had yellowish discoloration (14.0%), 49 patients had pain abdomen (49.0%),12 patients had itching (12.0%),3 patients had difficulty in breathing (3.0%),20 patients had decreased fetal movements (20.0%) and 22 patients had bleeding per vaginum /spotting (22.0%).

In control group,36 patients had pain abdomen (72%),1 patient had itching (2.0%),6 patients had decreased fetal movements (12.0%),3 patients had bleeding per vaginum/spotting (6.0%) as shown in Table 13,Figure 8

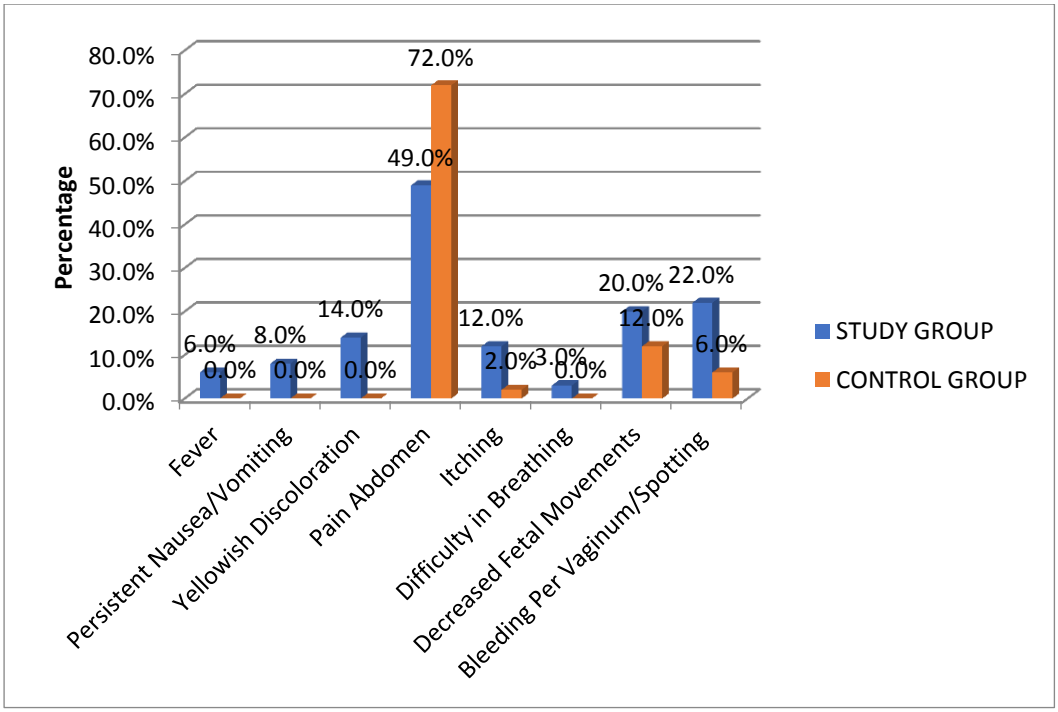

FIGURE 8- DISTRIBUTION OF STUDY-GROUP AND CONTROL GROUP ACCORDING TO SYMPTOMS

TABLE 14- DISTRIBUTION OF STUDY-GROUP AND CONTROL GROUP ACCORDING TO SIGNS

		STUDY GROUP		CONTROL GROUP		Total	Chi-square value	p-value
		No.	%age	No.	%age			
Pallor	No	85	85.0%	49	98.0%	134	5.912	0.022
	Yes	15	15.0%	1	2.0%	16		
Total		100	100.0%	50	100.0%	150		
Icterus	No	81	81.0%	50	100.0%	131	10.878	0.001
	Yes	19	19.0%	0	0.0%	19		
Total		100	100.0%	50	100.0%	150		
Hepatomegaly	No	94	94.0%	50	100.0%	144	3.125	0.179
	Yes	6	6.0%	0	0.0%	6		
Total		100	100.0%	50	100.0%	150		
Ascites	No	94	94.0%	50	100.0%	144	3.125	0.179
	Yes	6	6.0%	0	0.0%	6		
Total		100	100.0%	50	100.0%	150		
Increased BP readings	No	94	94.0%	49	98.0%	143	1.199	0.425
	Yes	6	6.0%	1	2.0%	7		
Total		100	100.0%	50	100.0%	150		

In our study,In study-group, 15 patients had pallor (15.0%),19 patients had icterus (19.0%),6 patients had hepatomegaly (6.0%),6 patients had ascites (6.0%) and 6 patients had increased BP readings (6.0%).In control group only 1 patient had pallor (2.0%) and 1 patient had increased BP readings (2.0%).p-value was 0.022 as shown in Table 9,Figure 9

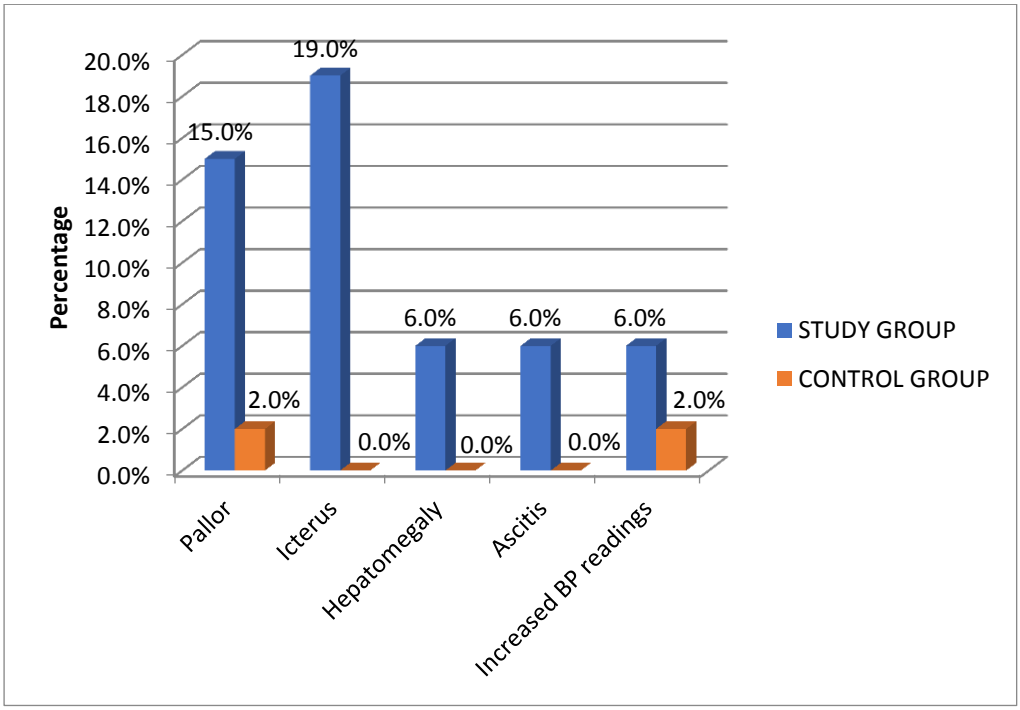

FIGURE 9- DISTRIBUTION OF STUDY-GROUP AND CONTROL GROUP ACCORDING TO SIGNS

INVESTIGATIONS

TABLE 15 - MEAN HAEMOGLOBIN (Hb) IN OUR STUDY

	STUDY GROUP		CONTROL GROUP		Z	p-value
	Mean	SD	Mean	SD		
Hb(gm/dl)	10.56	1.64	11.13	1.86	-2.855	0.004

In our study,Mean Haemoglobin was 10.56 gm/dl in Study group.Mean Haemoglobin in control group was 11.13 gm/dl.p-value was 0.004 as shown in Table 15,Figure 10

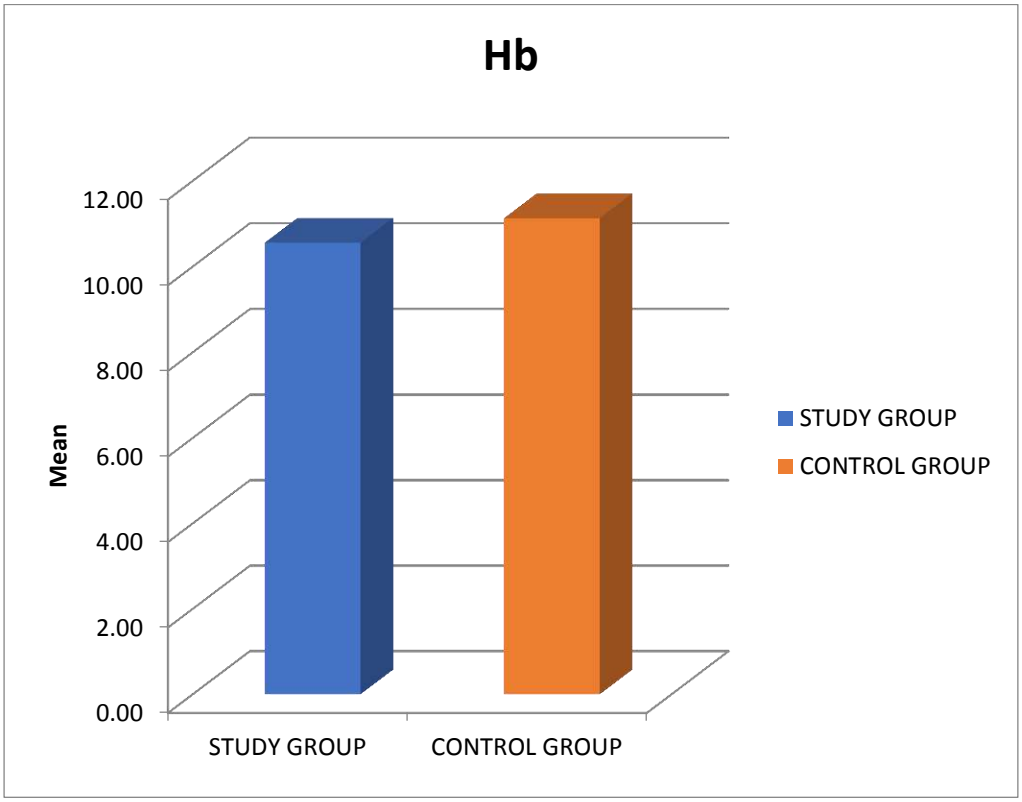

FIGURE 10- MEAN HAEMOGLOBIN (Hb) IN OUR STUDY

TABLE 16- MEANTLC IN OUR STUDY

	STUDY- GROUP		CONTROL GROUP		Z	p-value
	Mean	SD	Mean	SD		
TLC	12.12	5.09	10.39	2.75	-1.758	0.079

In our study,Mean TLC was 12.12 in Study-group.Mean TLC in control group was 10.39 .p-value was 0.079 as shown in Table 16,Figure 11

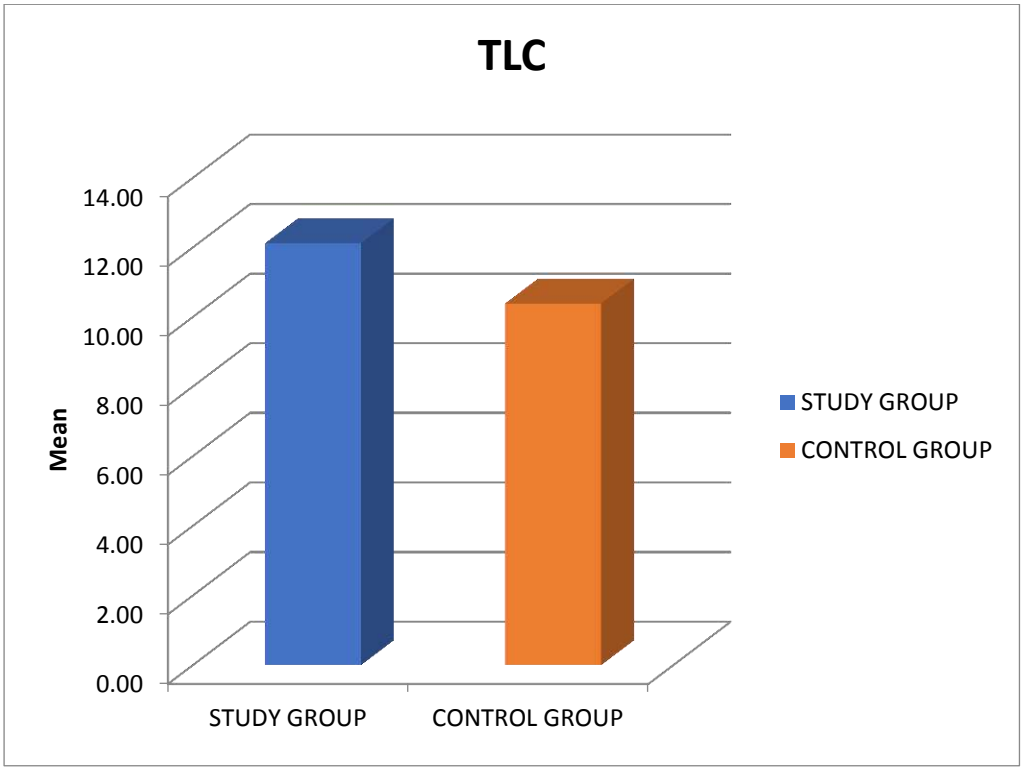

FIGURE 11- MEAN TLC IN OUR STUDY

TABLE 17 - MEAN PLATELET COUNT IN OUR STUDY

	STUDY GROUP		CONTROL GROUP		Z	p-value
	Mean	SD	Mean	SD		
Platelet count	184.95	74.60	206.28	64.51	-1.866	0.062

In our study, Mean Platelet count was 184.95 in Study-group.Mean platelet count in control group was 206.28 .p value was 0.082 as shown in Table 17,Figure 12

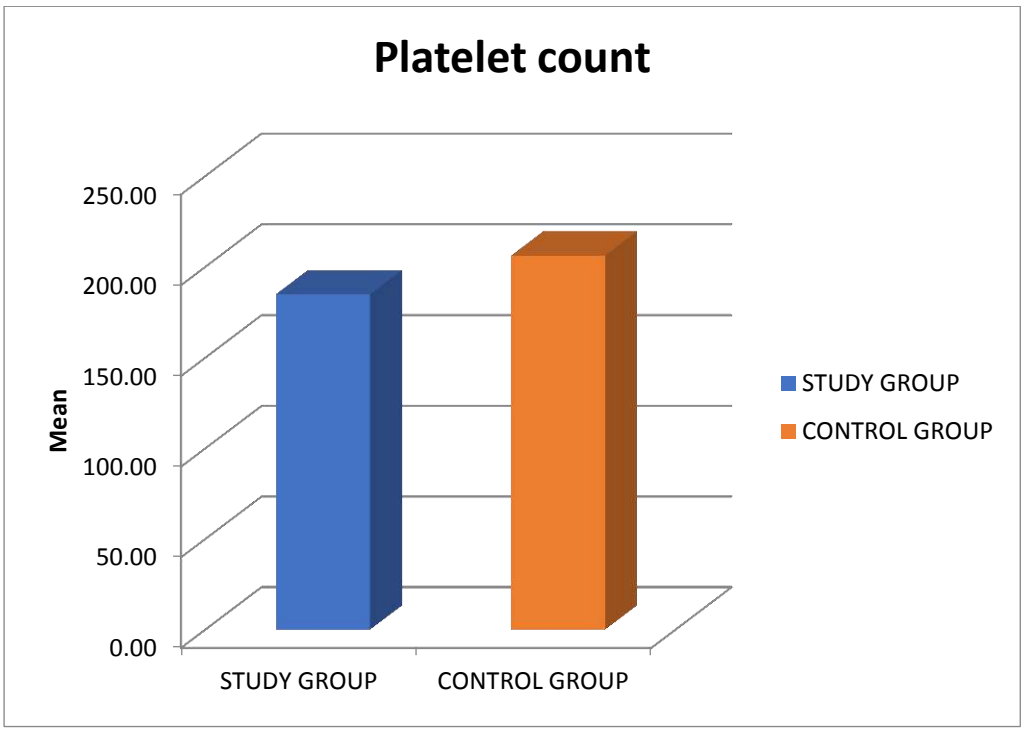

FIGURE 12- MEAN PLATELET COUNT IN OUR STUDY

TABLE 18 - MEAN LIVER ENZYMES IN OUR STUDY

	STUDY GROUP		CONTROL GROUP		Z	p-value
	Mean	SD	Mean	SD		
AST	274.00	451.12	33.80	17.47	-9.706	0.001
ALT	247.16	245.00	31.90	19.60	-9.702	0.001
ALP	262.51	174.33	151.12	92.10	-5.847	0.001

In our study,Mean AST was 274.00 ,Mean ALT was 247.18,Mean ALP was 262.51 in Study groups. Mean AST was 33.80 ,Mean ALT was 31.90,Mean ALP was 151.12 in patients enrolled in control group .p value was 0.001 as shown in Table 18,Figure 13

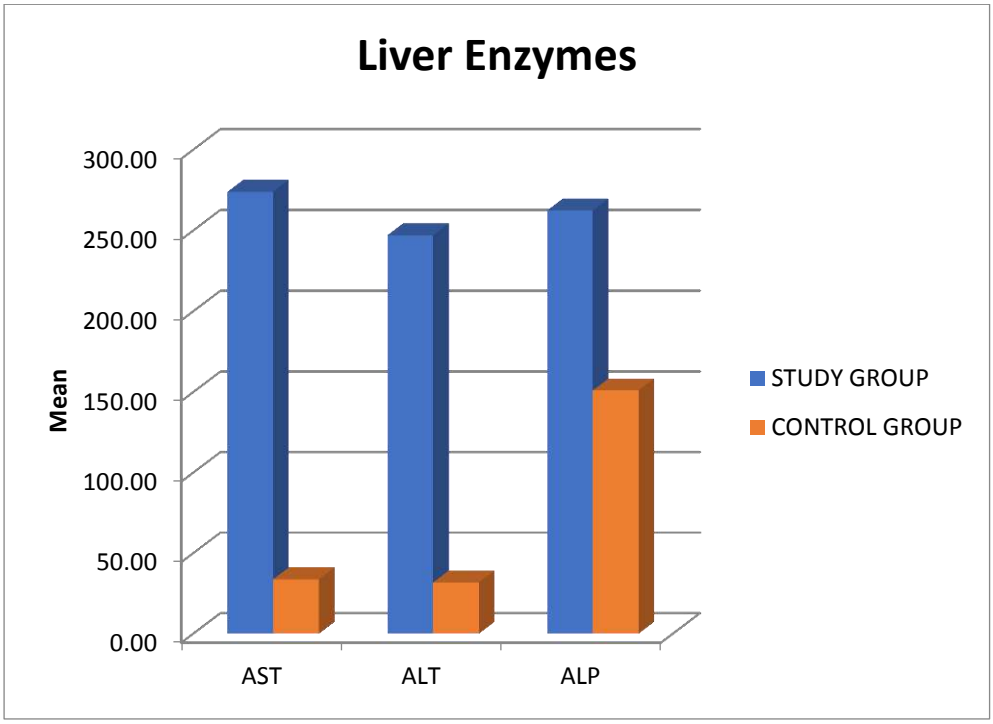

FIGURE 13- MEAN LIVER ENZYMES IN OUR STUDY

TABLE 19 - COMPARISON OF DISEASES IN STUDY-GROUP ACCORDING TO MEAN LIVER ENZYMES

	AST		ALT	
	Mean	**SD**	**Mean**	**SD**
ICP	332.96	505.633	287.49	254.398
Hyperemesis Gravidarum	122.00	41.581	151.33	79.198
Acute Liver Failure	1083.00	1234.608	844.00	642.053
HELLP	580.17	1061.119	367.67	454.884
Acute Fatty Liver of Pregnancy	950.33	1299.381	368.67	173.278
Pre eclampsia	199.40	125.401	244.60	144.024
Acute on Chronic Liver Failure	732.00	31.113	381.50	78.489
Drug Induced Liver Injury	171.50	54.447	254.50	191.626

In our study, In ICP, Mean AST was 332.96, Mean ALT is 287.39.In Hyperemesis Gravidarum, Mean AST was 122.00, Mean ALT was 151.33. In Acute Liver Failure, Mean AST was 1083.00, Mean ALT was 844.00. In HELLP, Mean AST was 580.17, Mean ALT was 367.67.In Acute Fatty Liver of Pregnancy, Mean AST was 950.33, Mean ALT was 368.67. In Pre eclampsia, Mean AST was 199.40, Mean ALT was 244.60. In Acute on Chronic Liver Failure, Mean AST was 732.00, Mean ALT was 381.50. In Drug Induced Liver Injury, Mean AST was 171.50, Mean ALT was 254.50 as shown in Table 19

TABLE 20- MEAN BILIRUBIN (TOTAL,DIRECT) IN OUR STUDY

	STUDY-GROUP		CONTROL GROUP		Z	p-value
	Mean	**SD**	**Mean**	**SD**		
Total Bilirubin	2.39	5.09	0.46	0.41	-4.474	0.001
D. Bilirubin	1.73	4.18	0.21	0.27	-4.401	0.001

In our study, mean Total Bilirubin was 2.39 ,mean Direct Bilirubin was 1.73 in Study group. mean Total Bilirubin was 0.46 and mean Direct Bilirubin was 0.21 in control group .p-value was 0.001 as shown in Table 20,Figure 14

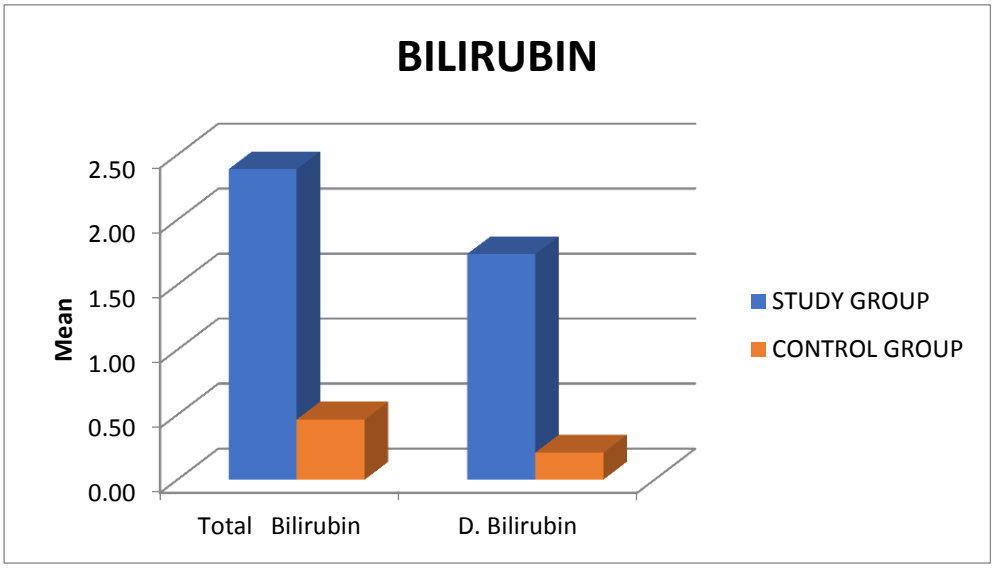

FIGURE 14 - MEAN BILIRUBIN (TOTAL, DIRECT) IN OUR STUDY

TABLE 21 - MEAN TOTAL PROTEIN AND ALBUMIN IN OUR STUDY

	STUDY GROUP		CONTROL GROUP		Z	p-value
	Mean	SD	Mean	SD		
Total Protein	6.26	0.8	6.31	0.49	-0.224	0.823
S. Albumin	3.21	0.47	3.63	0.34	-5.296	0.001

In our study, Mean Total Protein and Albumin was 6.26 and 3.21 in Study group and Mean Total Protein and Albumin was 6.31 and 3.63 in control group as shown in Table 21, Figure 15

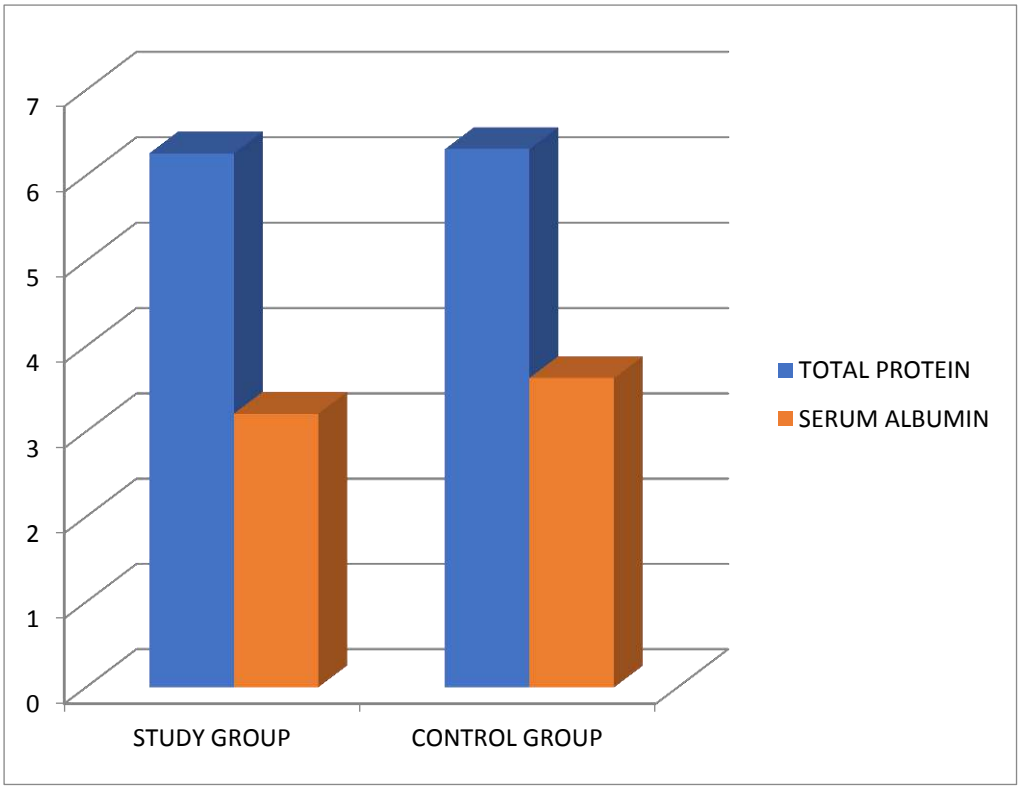

FIGURE 15- MEAN TOTAL PROTEIN AND ALBUMIN IN OUR STUDY

TABLE 22- MEAN OF BILE ACIDS IN OUR STUDY

	STUDY-GROUP		CONTROL GROUP		Z	p-value
	Mean	SD	Mean	SD		
Bile acids	57.90	69.35	12.38	4.92	-2.472	0.013

In our study,mean Bile acids was 57.90 in Study-group.In control group mean Bile acids was 12.38 with p-value of 0.013 as shown in Table 22

TABLE 23 - DISTRIBUTION OF STUDY-GROUP AND CONTROL GROUP ACCORDING TO MODE OF DELIVERY

		STUDY GROUP		CONTROL GROUP		Total	Chi-square value	p-value
		STUDY GROUP	STUDY GROUP	CONTROL GROUP	CONTROL GROUP			
Mode of delivery	**LSCS**	78	78.0%	15	30.0%	93	48.442	0.001
	Term NVD	12	12.0%	29	58.0%	41		
	Preterm NVD	6	6.0%	2	4.0%	8		
	Assisted Vaginal Delivery	0	0.0%	4	8.0%	4		
	Abortion	2	2.0%	0	0.0%	2		
	Classical C-Section	2	2.0%	0	0.0%	2		
Total		100	100.0%	50	100.0%	150		

In study-group, out of 100 patients, 78 patients delivered through LSCS (78.0%), 12 patients had Term Vaginal deliveries(12.0%), 6 patients had Preterm Vaginal deliveries (6.0%), 2 patients delivered through Classical C section (2.0%) and 2 patients had spontaneous abortion (2.0%).While In Control group, out of 50 patients, 29 had Term Vaginal

Deliveries(58.0%), 2 had Preterm Vaginal Deliveries(4.0%), 15 patients delivered through LSCS(30.0%) and 4 through Assisted Vaginal Delivery (8.0%).It was found that mode of delivery was significant factor in our study with p-value of 0.001 as shown in Table 23,Figure 16

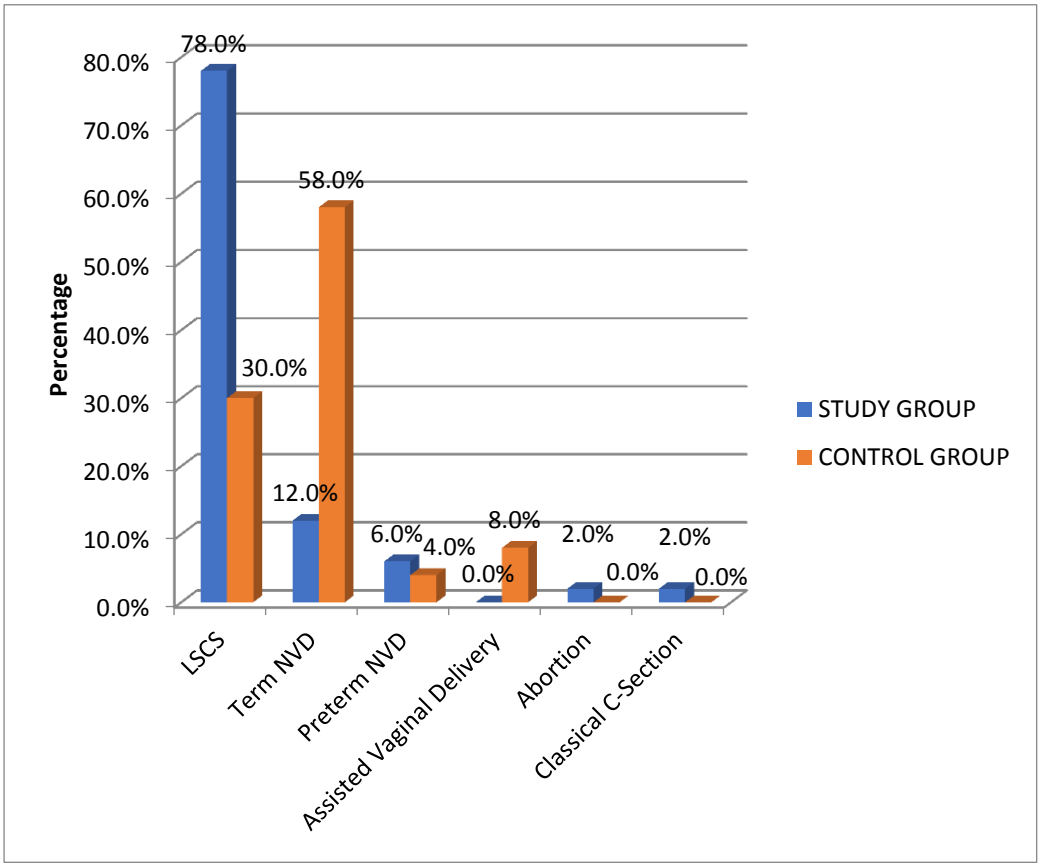

FIGURE 16- DISTRIBUTION OF STUDY-GROUP AND CONTROLGROUP ACCORDING TO MODE OF DELIVERY

TABLE 24 - DISTRIBUTION OF STUDY-GROUP AND CONTROL GROUP ACCORDING TO INDICATIONS OF CESAREAN SECTION

		STUDY GROUP		CONTROL GROUP		Total
		STUDY GROUP	STUDY GROUP	CONTROL GROUP	CONTROL GROUP	
Indications Of Cesarean Section	Fetal distress	34	34.00%	7	14.00%	41
	Scar tenderness	10	10.00%	1	2.00%	11
	Moderate ICP	7	7.00%	0	0.00%	7
	Previous LSCS	6	6.00%	1	2.00%	7
	Severe ICP	4	4.00%	0	0.00%	4
	Type IV placenta previa	4	4.00%	0	0.00%	4
	Deteriorating Maternal condition	3	3.00%	0	0.00%	3
	Mild ICP	3	3.00%	0	0.00%	3
	APH	0	0.00%	2	4.00%	2
	ACLF	2	2.00%	0	0.00%	2
	Failure of induction	2	2.00%	0	0.00%	2
	Pre eclampsia	2	2.00%	0	0.00%	2
	Acute fatty liver of pregnancy	1	1.00%	0	0.00%	1
	Arrest of descent of head in 2nd stage of labour	1	1.00%	0	0.00%	1
	Atonic PPH	1	1.00%	0	0.00%	1
	Decreased Fetal movements	0	0.00%	1	2.00%	1
	Unfavourable cervix	1	1.00%	0	0.00%	1
Total		100	100.00 %	50	100.00%	150

In our study, Indication of caesarean section in study group was fetal distress in 34 patients (34.0%),scar tenderness in 10 patients (10.0%),moderate ICP in 7 patients (7.0%),previous LSCS in 6 patients (6.0%),severe ICP in 4 patients (4.0%),Type IV placenta previa in 4 patients (4.0%),Deteriorating Maternal condition in 3 patients (3.0%),mild ICP in 3 patients (3.0%),Acute on Chronic Liver Failure (ACLF) in 2 patients (2.0%),failure of induction in 2 patients(2.0%),pre-eclampsia in 2 patients (2.0%), Acute fatty liver of pregnancy in 1 patient (1.0%),Arrest of descent in 2nd stage of labour in 1 patient (1.0%),Atonic PPH in 1 patient (1.0%).While In Control group,Indication of caesarean section was fetal distress in 7 patients (14.0%), scar tenderness in 1 patient (2.0%), previous LSCS in 1 patient (2.0%),APH in 2 patients (4.0%) and Atonic PPH in 1 patient (2.0%) as shown in Table 24,Figure 17

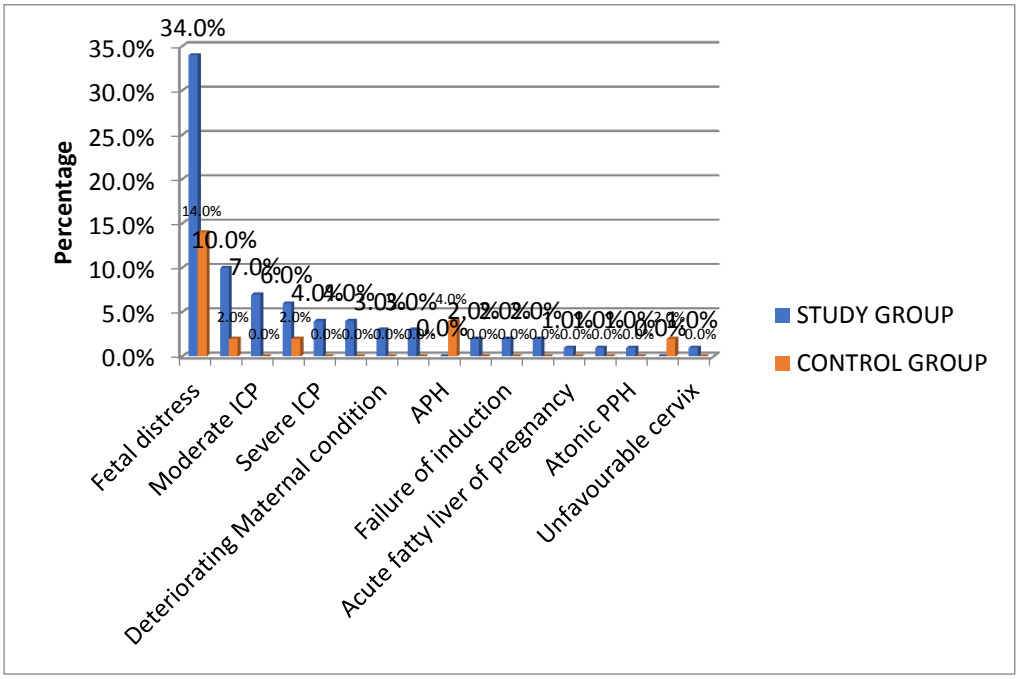

FIGURE 17 - DISTRIBUTION OF STUDY-GROUP AND CONTROL GROUP ACCORDING TO INDICATIONS OF CESAREAN SECTION

TABLE 25 - DISTRIBUTION OF STUDY-GROUP AND CONTROL GROUP ACCORDING TO FETAL GENDER

		STUDY GROUP		CONTROL GROUP		Total	Chi-square value	p-value
		STUDY GROUP	STUDY GROUP	CONTROL GROUP	CONTROL GROUP			
Fetal GENDER	**Female**	44	44.0%	20	40.0%	64	0.135	0.714
	Male	58	58.0%	30	60.0%	88		

In our study,In Study-group,44 females and 58 males were born.In control group,20 females and 30 males were born as shown in Table 25,Figure 18

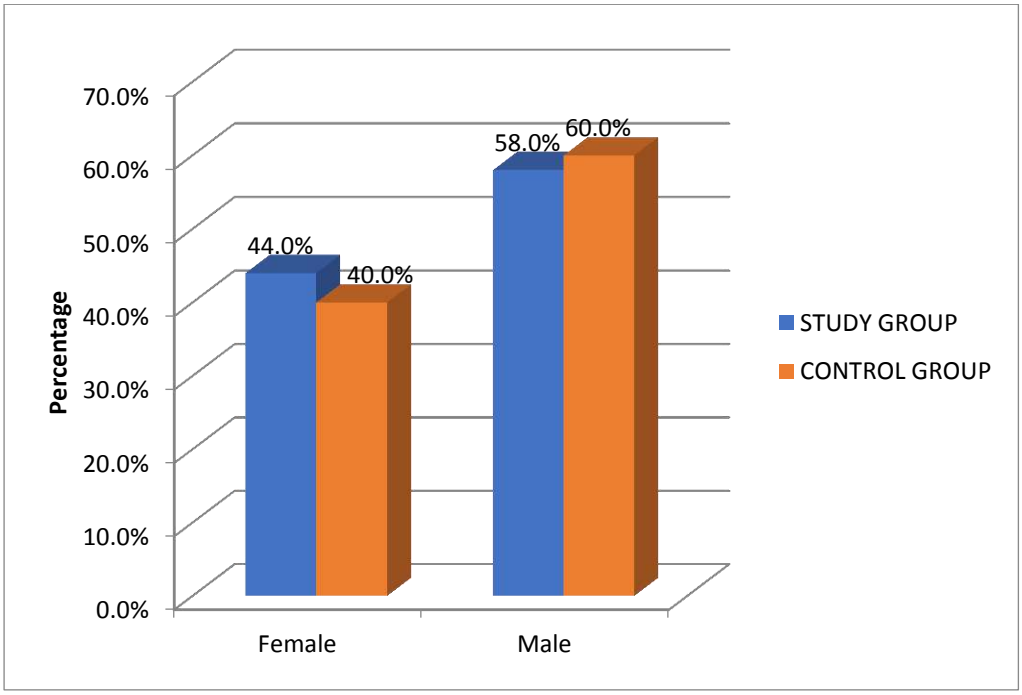

FIGURE 18 - DISTRIBUTION OF STUDY-GROUP AND CONTROL GROUP ACCORDING TO FETAL GENDER

TABLE 26 - DISTRIBUTION OF STUDY-GROUP AND CONTROL GROUP ACCORDING TO FETAL BIRTH WEIGHT

		STUDY GROUP	STUDY GROUP	CONT-ROL GROUP	CONT-ROL GROUP			
Fetal Birth Weigh t (kg)	< 1.5	21	21.0%	2	4.0%	23	24.065	0.001
	1.5-2	5	5.0%	1	2.0%	6		
	2.-2.5	37	37.0%	8	16.0%	45		
	2.5-3	24	24.0%	21	42.0%	45		
	> 3	13	13.0%	18	36.0%	31		
Total		99	99.0%	50	100.0%	149		

In our study,In study-group,out of 100 deliveries ,21 babies had fetal weight < 1.5 kg (21.0%), 5 babies had fetal weight in between 1.5-2 kg (5.0%),37 babies had fetal weight in between 2-2.5 kg (37.0%), 24 babies had fetal weight in between 2.5-3 kg (24.0%), 13 babies had fetal weight >3 kg (13.0%).In Control group out of 50 deliveries, 2 babies had fetal weight in between < 1.5 kg (4.0%),1 baby had fetal weight in between 1.5-2 kg (2.0%), 8 babies had fetal weight in between 2-2.5 kg (16.0%), 21 babies had fetal weight in between 2.5-3 kg (42.0%),18 babies had fetal weight >3 kg (36.0%) as shown in Table 26 ,Figure 19

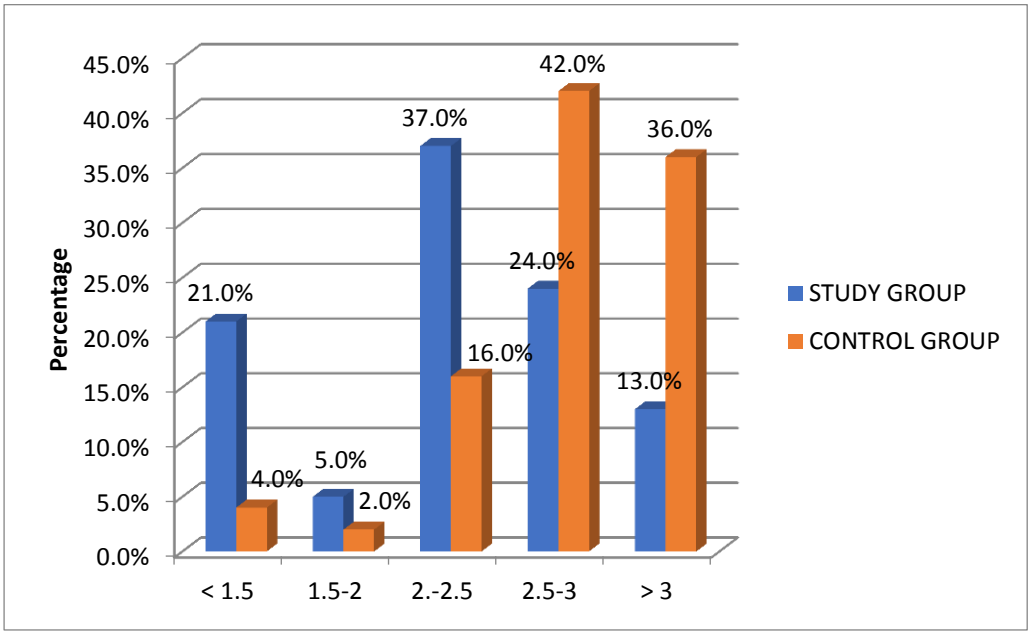

FIGURE 19 - DISTRIBUTION OF STUDY-GROUP AND CONTROL GROUP ACCORDING TO FETAL BIRTH WEIGHT

TABLE 27 – MEAN FETAL BIRTH WEIGHT OF STUDY-GROUPAND CONTROL GROUP

	STUDY GROUPS		CONTROL GROUP		Z	p-value
	Mean	SD	Mean	SD		
Fetal Weight	2.20	0.79	2.81	0.57	-4.999	0.001

Mean fetal birth weight was 2.2 kg in Study-group While in Control group mean fetal birth weight was 2,81 kg with p-value was 0.001. It was found that fetal birth weight was a significant factor in our study in Table 27, Figure 20.

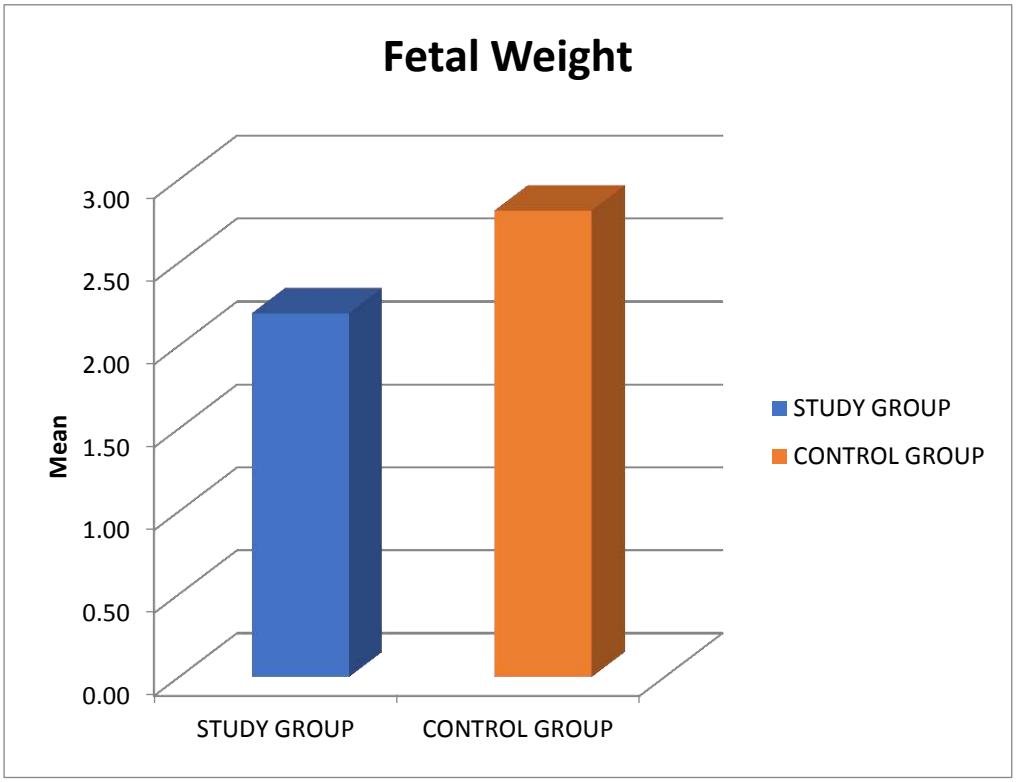

FIGURE 20 – MEAN FETAL BIRTH WEIGHT OF STUDY-GROUP AND CONTROL GROUP

TABLE 28 - DISTRIBUTION OF STUDY-GROUP AND CONTROL GROUP ACCORDING TO FETAL OUTCOME

		STUDY GROUP		CONTROL GROUP		Total	Chi-square value	p-value
		STUDY GROUP	STUDY GROUP	CONTROL GROUP	CONTROL GROUP			
Fetal Outcome	**Normal Live Birth**	48	48.0%	47	94.0%	95	34.632	0.001
	Preterm birth	22	22.0%	1	2.0%	23		
	Intrauterine Death	14	14.0%	1	2.0%	15		
	Fetal Growth restriction	12	12.0%	0	0.0%	12		
	Abortion	4	4.0%	0	0.0%	4		
	Stillbirth	0	0.0%	1	2.0%	1		
Total		100	100.0%	50	100.0%	150		

In our study, out of 100 cases regarding fetal outcome, 48 had normal live birth (48%),22 had pre term birth (22%),14 presented with intrauterine death (14%),12 had fetal growth restriction (12%) and 4 abortions were there (4%).While In Control group, 47 babies had normal live birth (94%), 1 had pre term birth (2%) ,1 had intrauterine death (2%) and 1 still birth was there (2%).It was found that fetal outcome was a significant factor in our study with p-value of 0.001 as shown in Table 27 ,Figure 21

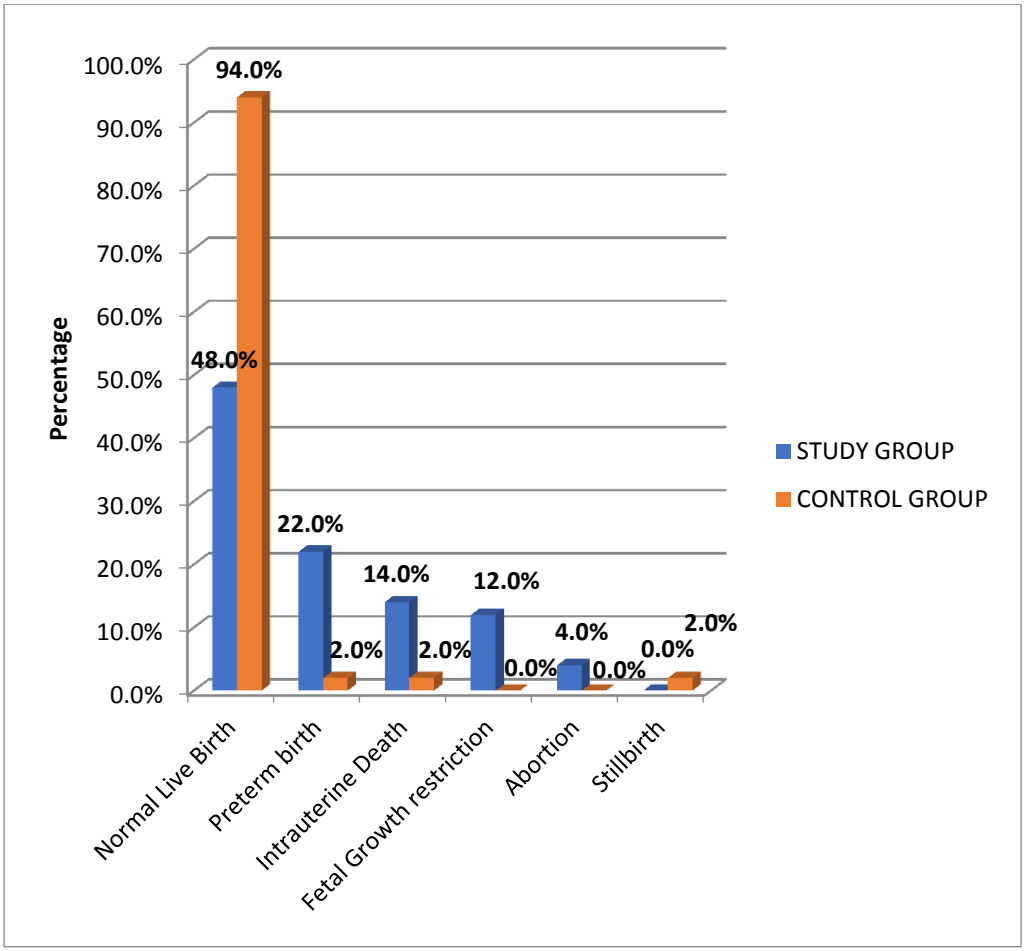

FIGURE 21 - DISTRIBUTION OF STUDY-GROUP AND CONTROL GROUPACCORDING TO FETAL OUTCOME

Table 29 -DISTRIBUTION OF STUDY-GROUP AND CONTROL GROUP ACCORDING TO FETAL COMPLICATIONS

		Study Group		Control Group		Total	Chi-square value	p-value
		Study Group	Study Group	Control Group	Control Group			
Feta complications(If Any)	**No**	62	62.0%	50	100.0%	112	25.446	0.001
	Prematurity	30	30.0%	0	0.0%	30		
	NICU admission	6	6.0%	0	0.0%	6		
	Perinatal Asphyxia	2	2.0%	0	0.0%	2		
Total		100	100.0%	50	100.0%	150		

In our study, 62 babies had no fetal complication (62%),30 babies were born prematurely (30%),6 required NICU admission(6%) and 2 had perinatal asphyxia (2%).While In control group, there were no complications noted. p-value was 0.001 as shown in Table 29,Figure 22

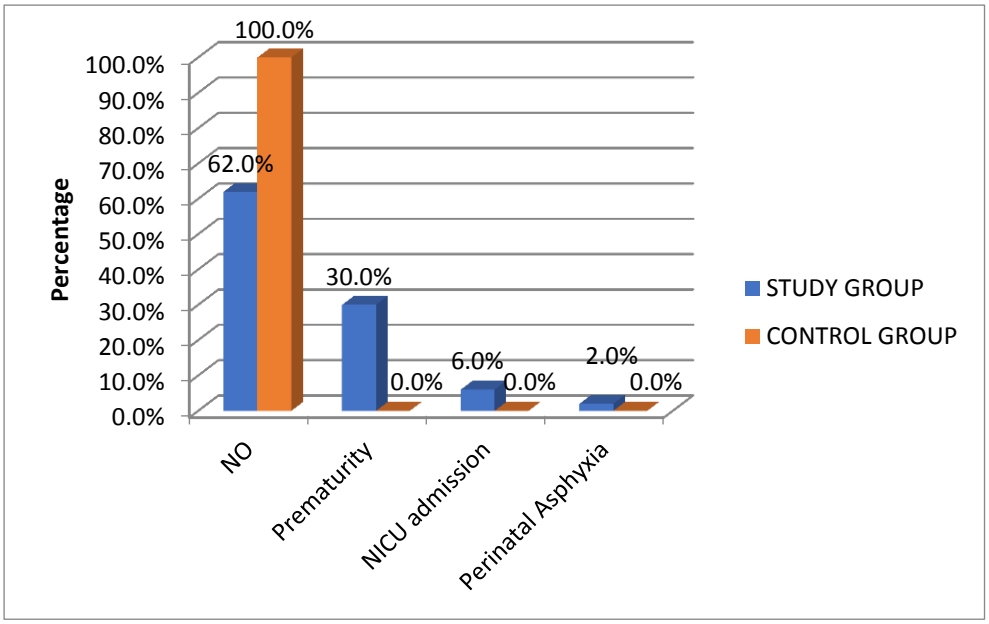

FIGURE 22– DISTRIBUTION OF STUDY-GROUP AND CONTROL GROUP ACCORDING TO FETAL COMPLICATIONS

TABLE 30 - DISTRIBUTION OF STUDY-GROUP AND CONTROL GROUP ACCORDING TO MATERNAL OUTCOME

		Study Group		Control Group		Total	Chi-square value	p-value
		Study Group	Study Group	Control Group	Control Group			
Maternal Outcome	Normal	57	57.0%	49	98.0%	106	27.039	0.001
	Prolonged Hospital stay	40	40.0%	1	2.0%	41		
	Multiorgan failure	2	2.0%	0	0.0%	2		
	Mortality	1	1.0%	0	0.0%	1		
Total		100	100.0%	50	100.0%	150		

In our study, In study-group,57 females had normal outcome (57.0%),40 had prolonged hospital stay (40.0%),2 had multiorgan failure (2.0%) and 1 maternal mortality (1.0%) was there. While In Control group, 49 females had normal outcome (98.0%) and 1 had prolonged hospital stay (2.0%).There was no patient with multiorgan failure or mortality in Control group. Maternal outcome was a significant factor with p-value of 0.001 a shown in Table 30 ,Figure 23

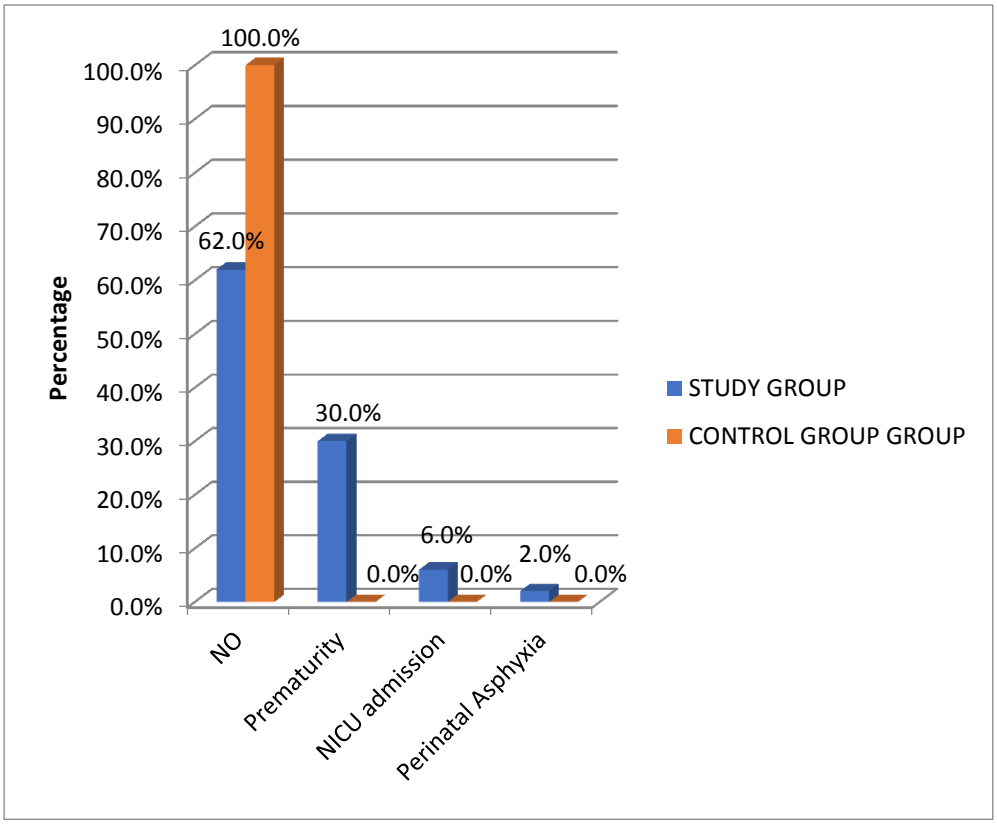

FIGURE 23 - DISTRIBUTION OF STUDY-GROUP AND CONTROL GROUP ACCORDING TO MATERNAL OUTCOME

TABLE 31 - DISTRIBUTION OF STUDY-GROUP ACCORDING TO MATERNAL COMPLICATIONS

	STUDY-GROUP	No. of Cases	%age
Maternal Complications (If Any)	**No**	71	71.0%
	Sepsis	8	8.0%
	HELLP	6	6.0%
	Pre -eclampsia	5	5.0%
	Acute Liver Failure	5	5.0%
	Anemia	4	4.0%
	APH	3	3.0%
	Shock	3	3.0%
	Obstetrical Hysterectomy	3	3.0%
	Acute kidney injury	2	2.0%
	DIC	1	1.0%
	Pulmonary edema	1	1.0%

In our study, among maternal complications,8 patients had sepsis (8%) out of which 3 patients (3%) had septic shock,6 patients had HELLP (6%).5 patients had pre-eclampsia (5%),5 patients had acute liver failure (5%),4 patients had anemia (4%),3 patients had APH (3%), 3 patients had obstetrical hysterectomy (3%), 2 patients had acute kidney injury (2%), 1 patient had DIC (1%) and 1 patient had pulmonary edema (1%) as shown in Table 31 ,Figure 24

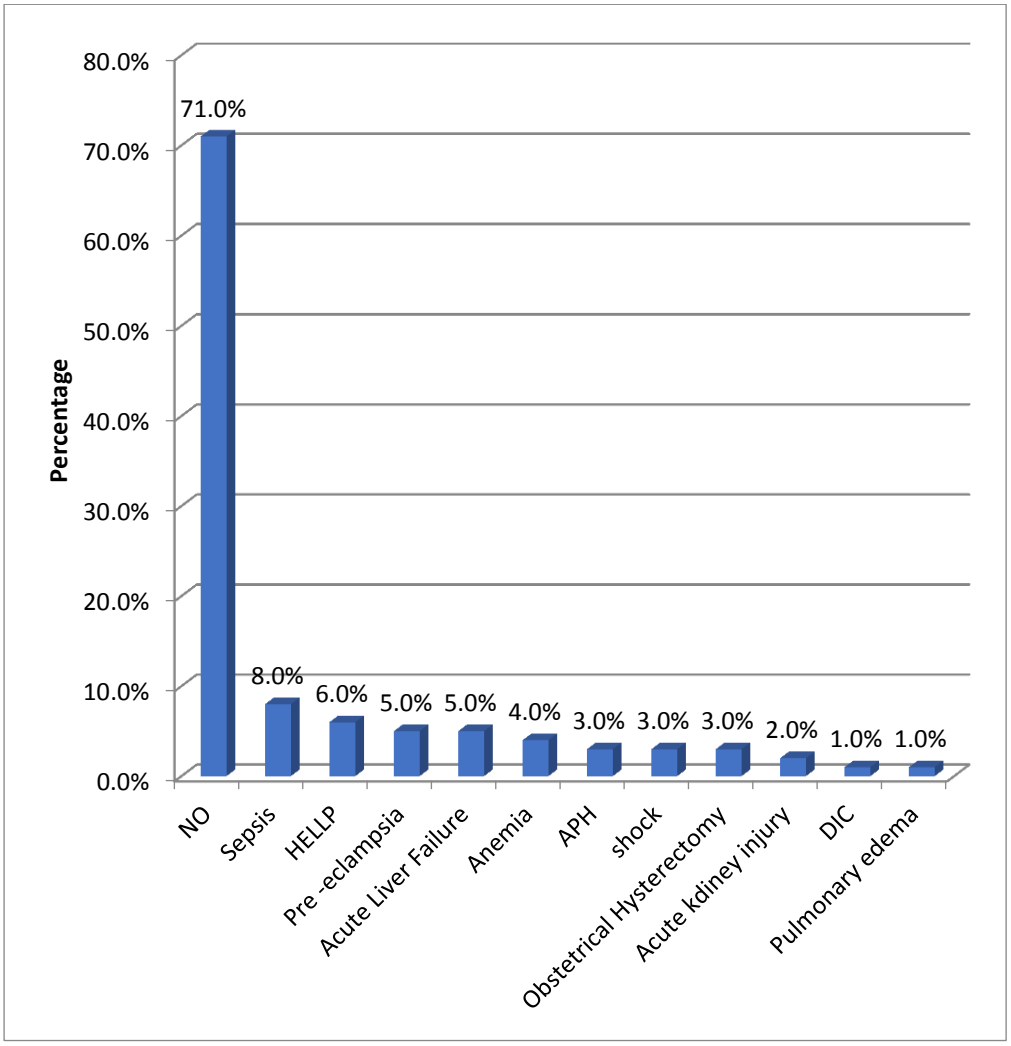

FIGURE 24 - DISTRIBUTION OF STUDY-GROUP ACCORDING TO MATERNAL COMPLICATIONS

DISCUSSION

Liver disease during pregnancy presents a significant concern, impacting approximately 3% of pregnancies and carrying risks for both mothers and babies. A prospective study was conducted at the Medicine and Gynaecology department at the tertiary care centre, Dayanand Medical College and Hospital in Ludhiana. This study involved 100 pregnant females diagnosed with liver disease and 50 control pregnant females without liver disease, who were admitted in the hospital over a period of 15 months from September 15, 2022, to December 15, 2023. The primary objective of this study was to analyze the clinical profile of liver disease in hospitalized pregnant women and to compare the maternal-fetal outcomes among them.

In our study, the highest number of patients were within the 26-30 years age group, with 36 patients (36%) among Study group and 40 patients (40%) among control group. Meena.N.satia et al. [110] 2016 reported 45.4% of the patients were within 25-29 years age group. This could also be, because of the fact that the majority of reproductive-aged women in our Hospital are within 26-30 years age group.

This age group is different from previous studies, such as Sharma et al.[111] (2013), where the peak incidence was in the 21–25 years age group, and Jain et al.[112] (2014), which reported a mean age of 25.4 years. Tiwari et al [113]. found that 68.69% of patients were aged 20-29 years, and Kumar Patra et al. [114] noted that 44.4% of patients were aged 21–25 years. Despite these variations, our study did not identify age as a significant risk factor, as indicated by a p-value of 0.184 for a sample size of 100.

Regarding the incidence of liver diseases during pregnancy in our hospital, there were 280 cases of deranged liver function tests (LFTs) out of 1480 deliveries during a 15-month period. From these, 100 cases that met the inclusion criteria were included in the study. The

incidence of liver diseases during pregnancy was found to be 18% in our study. A.Mahajan et al. (2023)[108] reported a high prevalence of about 4.1%.The elevated incidence observed in our study is likely because our hospital is a tertiary care centre, receiving numerous referrals and handling many high-risk pregnancies. Furthermore, the overall number of deliveries performed is relatively low.

Among other such studies conducted, Sarkar et al. (1992)[115] reported an incidence of 0.23%.Umang Rathi et al**.** (2007)[67] reported an incidence of 0.9% out of total 12091 pregnancies. Neema et al. (2013) [116]in Wardha reported an incidence of 0.4% over six years. Krishnamoorthy et al. (2016) [117] found an incidence of 0.29%.A 2016 study at Seth Gordhandas Sunderdas Medical College [111] reported a higher incidence of 0.81% and S.N. Vinayachandran et al. (2020)[118]reported an incidence of 0.22% of liver diseases resulting in complicated pregnancies. The low incidence in these studies could be an underestimate because many women arrived at the hospital solely for delivery, being referral, without prior antenatal visits. Furthermore, we did not perform biochemical liver function tests on women without symptoms.

These findings illustrate that the incidence of liver diseases in pregnancy varies significantly, not only between developing and developed countries but also within different regions of the same country. This variability can be attributed to differences in healthcare systems, access to medical services, socioeconomic factors, regional health conditions and screening programs.

In our study of 100 patients, 62 patients (62%) had pregnancy-specific liver disorders,22 patients (22%) had liver disorders unrelated to pregnancy, and In 16 patients (16%) no definitive cause could be determined for the abnormal liver function tests. Similarly in a study done by Umang Rathi et al[67].fifty six (52.3%) had pregnancy-specific liver disorders out of 12061 deliveries.

ICP was the most prevalent liver disorder among pregnancy-specific liver disorders in our study, affecting 45 patients out of the 62 (72%): in which 20 patients had mild ICP, 17 patients had moderate ICP, and 8 patients had severe ICP. The second most common pregnancy-specific liver disorder was HELLP syndrome, with 6 cases (9.6%), followed by pre-eclampsia with 5 cases (8%), while both AFLP and Hyperemesis Gravidarum had 3 cases each (5%).The similar findings with ICP as most prevalent liver disorder in pregnancy was reported by A. Mahajan et al. (2023) [108] in which out of 940 pregnant females studied, the prevalence of ICP was most common with 588 cases (62.5%). The same findings were reported in study done by Yannik et al. [119]& Dhawan and Sainani et al.[120]

Contrary to this,S.A.Panchbudhe et al.(2022) [121] reported in his study that HELLP syndrome was the most frequent (80.8%)followed by pre-eclampsia (19.2%). Tiwari et al[113] reported HELLP syndrome as most common liver specific disorder during pregnancy occurring in 15.62% of patients in their study. In a study by Jain et al.[112], HELLP syndrome was observed in 27.2% of 55 patients. S.N.Vinayachandran[118] reported HELLP syndrome was the commonest cause of liver disorders followed by viral hepatitis. Of the 52 Study groups studied in his study, 18 (34.6%) were due to HELLP syndrome, and 17 (32.7%) were due to viral hepatitis. Tank et al.[122] also reported that most of the patients (80.7%) had HELLP syndrome.

In our study, Out of 22 patients (22%) among liver disorders unrelated to pregnancy, tropical fever was the most common, accounting for 11 (50%)cases, including dengue (7 patients), leptospirosis (3 patients), and scrub typhus (1 patient). Viral hepatitis accounted for 5 (22.7%) cases, specifically Hepatitis E (4 patients) and Hepatitis A (1 patient). There were 2 cases each of Acute Liver Failure, Acute on Chronic Liver Failure, and Drug-Induced Liver Failure (caused by progesterone).

Earlier studies by Meena et al.[110] and Neema et al.[116] identified viral hepatitis as the most prevalant cause of jaundice in pregnancy.

Similarly Krishnamoorthy et al[117]. reported 51%, Shukla et al.[123] reported 57% and Harshad et al. [124] reported 47% of cases of viral hepatitis in their respective studies.

Our findings suggest a recent decline in the incidence of viral hepatitis, likely due to increased public awareness, vaccination and improved hygiene practices, indicating better preventive measures and healthcare practices that reduce the spread of viral infections.

Regarding Gravida distribution, In our study of 100 sample size,59 patients (59%) were multigravida while remaining 41 patients (41%) were primigravida. Among the control group,35 patients (70%) were multigravida and remaining 15 (30%) were primigravida. Similarly Suri DA et al.(2014) [125] reported in their study out of total 50 pregnancies with jaundice, 28 (56%) patients were multiparous & 22 (44%) were nulliparous.In a study conducted by S.A. Panchbudhe et al. (2022)[121] involving 120 patients, 52.5% were multigravida and 47.5% were primigravida. On the other hand, in a study done by Singh et al.[126] found that 72.9% of Study groups were primigravida. AFLP, pre-eclampsia, and HELLP syndrome were more commonly observed in multiparous women in our study whereas Intrahepatic Cholestasis of Pregnancy, Viral hepatitis, and Hyperemesis Gravidarum were more common in primigravida patients. These findings align with previous studies done by Jain et al.[112]

According to Gestational age distribution, Out of 100 patients, maximum number of patients 45 (45%) had gestational age > 36 weeks while In the control group out of 50 patients,42 patients (84%) had gestational age> 36 weeks. Mean gestational age was 32 weeks in study group and 36 weeks in control group in our study. A p-value of 0.001 signifies a statistically significant difference in gestational ages between the two groups in our study, supporting the conclusion that liver disorders in pregnancy are linked to preterm deliveries.

Coming to Trimester wise distribution, In our study out of 100, the majority of patients 80 patients (80%) presented in the 3rd trimester of pregnancy, with 49 presenting at term.16 patients (16%) presented in their 2nd trimester, and 4 patients (4%) presented during the 1st trimester. Similar findings were found in a study done by Singh et al.[126] where 90.59% of Study groups presented in the 3rd trimester of pregnancy. It was found that trimester was a significant risk factor in our study with p- value of 0.011.

Liver diseases exhibited a specific pattern of association with the duration of pregnancy. Out of 45 cases of ICP, 38 presented in 3rd trimester (84%) and 7 presented in 2nd trimester (15%). Most of the cases of ICP were observed in the 3rd trimester of pregnancy similar to the study by Ch'gh et al[1] in South Wales.

Out of 6 HELLP cases in our study,4 presented in 3rd trimester (66%) and 2 presented in 2nd trimester (33%).All 5 cases of pre-eclampsia and acute viral hepatitis presented in the 3rd trimester (100%) while out of 3 cases of hyperemesis gravidarum ,2 presented in the 1st trimester (66%) and 1 came in the 2nd trimester (33%). This trimester oriented presentation is comparable with previous studies done by Tiwari et al[113] and Jain et al.[112]

Distribution of patients according to symptoms in our study,49 patients presented with pain abdomen (49.0%),14 patients had yellowish discoloration(14.0%),12 patients had pruritus (12.0%) and 8 patients presented with nausea and vomiting (8.0%).Similarly in a study done by Meena.N.Satia (2016)[111]maximum number of patients (58%) came with complaints of jaundice with abdominal pain followed by pruritus (31%).In a study done by Swati Sharma et al.[111]Pruritus was most common presenting symptom present in 60% of patients.

In our study, Patients presented with decreased fetal movements and Bleeding per vaginum were 20% and 20% respectively While Fever and breathing difficulty at presentation was seen in only 6% and 3% patients respectively. While in control group, no patient had jaundice and only 1 patient complained of itching. Jaundice and itching were found to be significant factors among symptoms in our study with p-value of 0.005.

Distribution of patients according to Liver function tests in our study, among bilirubin levels, mild bilirubin elevation (<6 mg/dl) was observed in 20patients (20%), while 5 patients had moderate elevation in bilirubin (6-15mg/dl) (5%).Severe bilirubinemia>15mg/dl) was detected in 4patients (4%), out of which 3 patients exhibiting levels exceeding 20 mg/dl.

In a study done by Krishnamoorthy et al.[117], bilirubin levels varied widely between 2.8 mg/dL and 18.4 mg/dL, with 7.84% of patients having high serum bilirubin (>15 mg/dl).In a study done by N.tripti et al.[127]Serum billirubin was > 10 mg/dl in 48.48% of the women.

Among liver enzymes, Most patients in our study had AST (SGOT) and ALT (SGPT) levels below 200 U/L (68% and 61%, respectively). AST (SGOT) and ALT (SGPT) levels between 200-500 U/L were seen in 25% and 28% respectively. In a study on feto-maternal outcomes in jaundice during pregnancy by Nagaria et al. [128], 75% patients had SGOT/SGPT levels below 500 U/L, which aligns with similar findings reported by Krishnamoorthy et al.[117], where 93% of patients had an SGPT value below 500 U/L. Additionally, only 5 patients showed SGOT values exceeding 1000, while 3 patients had SGPT values exceeding 1000 U/L.

Total protein were normal in 68 patients (68%), with 24 patients in the study group showing levels between 5–5.9 g/dL, and 7 patients having a total protein value below 5 g/dL. While in a study done by S.N.Vinayachandran et al[118]. The total protein were normal in 23.1%. A total protein level of 5–5.9 g/dL was seen in 61.5% of patients and 15.4% had a total protein value of less than 5 g/dL.

Among the 100 study group patients analyzed, 20 underwent vaginal delivery (20%) with 12 reaching full term and 8 delivering prematurely. Conversely, 78patients underwent cesarean sections (CS) (78%), with 2 being classical cesarean sections (CCS) and the remainder being lower segment cesarean sections (LSCS). The primary indication for CS was fetal distress in 34 patients, followed by ICP in 14 patients, out of which 3 were mild, 7 were moderate, and 4 were severe. Additionally, 16 patients had a history of previous CS, with 10 of them experiencing scar tenderness. Placenta previa type IV was observed in 4 patients. Three CSs were performed in view of deteriorating maternal condition. One CSs was conducted due to arrest of descent, while 2 cases were attributed to failed induction and pre-eclampsia. Two CSs were related to ACLF, and one each was due to AFLP and an unfavourable cervix.

In a study conducted by Krishnamoorthy et al.[117] 2.7% of the deliveries were vaginal, while 21.56% were lower segment cesarean sections (LSCS). On the other hand, Satia's study reported that 69% of the deliveries were vaginal.

The increased number of cesarean sections in our study can be explained by the fact that our hospital being a tertiary care centre receives a high number of referrals from other hospitals, mainly involving high-risk pregnancies and presenting mostly during labour.
Maternal mortality and complications were thoroughly evaluated among the 100 patients. The majority of 71 patients experienced no complications, while 29 patients encountered various issues. The most

common complication was sepsis with 8 patients (8%) out of which 3 cases progressed to septic shock necessitating High end antibiotics, Inotropic support and ICU admission. Additionally, 2 patients (2%) developed acute kidney injury requiring dialysis, 1 patient (1%) experienced disseminated intravascular coagulation (DIC), and 4 patients (4%) had anemia requiring blood transfusions. An association was found between HELLP syndrome and complications such as pulmonary edema, antepartum hemorrhage (APH), sepsis, and acute kidney injury. Among the groups with APH, three required obstetrical hysterectomy, and one was associated with wound sepsis. Complications related to HELLP syndrome included DIC, pleural effusion, and pulmonary edema.

Maternal outcomes in the study group were significant with 57% cases results being normal while 40% patients had Prolonged hospital stay. Multi organ Failure was reported in 2% cases. There was one case of maternal mortality (1%), which was associated with Acute Fatty Liver of Pregnancy. The patient presented in the first trimester with symptoms of nausea and vomiting, along with jaundice, ascites, and hepatomegaly. She also developed coagulopathy and hepatic encephalopathy. Despite a premature delivery and transfer to the Gastroenterology department, her condition worsened rapidly, resulting in acute hepatic failure and ultimately her demise.

On the other hand in our study in control group 98% mothers had normal outcome, only 1 case (2%) had Prolonged hospital stay.

Similarly Swati Sharma et al. reported 1 case of maternal mortality in their study out of 30 patients.In the study done by Krishnamoorthy et al. [[117],] 4 maternal deaths were reported out of 51 cases. The causes included AFLP in two cases, HELLP syndrome in one case, and rupture of esophageal varices in another case. Similarly, the study done by Pranatti Mitta [129] reported a maternal mortality rate of 4.76%. Maternal mortality in pregnancy with liver disease has dropped to 0%-

1.1% in recent studies from the West .Preterm deliveries were significantly higher, accounting for 69.2% compared to term deliveries at 30.7%, which is consistent with the findings from our study. In our study, out of 100,the total number of preterm deliveries were 22 (22%),48 (48%) were term deliveries whereas In control group out of 50 deliveries, 47 (94%) were term and only 1 preterm delivery was there (2%).This shows a higher incidence of preterm deliveries in pregnancies complicated by liver disorders when compared to the general population. A study by Kumar et al.[114]also showed a higher incidence of pre term deliveries.

When analyzing the birth weight distribution in our study, A significant proportion of babies in the study group were born with low birth weights with 21% weighing < 1.5 kg ,42% weighing < 2.5 kg and 37% > 2.5 kg.On the other hand, the control group had lower percentages with 4% weighing <1.5 kg and 18% < 2.5 kg indicating a higher prevalence of low birth weights in the study population. These findings correspond with those of Mitta's [129] study. Kumar Patra et al. [114]reported 3 patients (2.1%) had very low birth weight < 1.5 kg , 88 patients (61.1%) had low birth weight <2.5 kg, and 53 (36.8%) patients had normal birth weight >2.5 kg.

Regarding Fetal outcomes in our study, 48 (48%) had normal live births, 22(22%) neonates were delivered prematurely and 12(12%) had fetal growth restriction.14 (14%) Intrauterine deaths and 4 (4%) spontaneous abortions were reported.6 (6%) required Neonatal ICU admission and perinatal asphyxia was reported in 2(2%) neonates. Whereas in control group 47 neonates (94%) had normal live birth and only 1 (2%) preterm was reported. There was 1 Intrauterine death (2%) and 1 stillbirth (2%) reported in control group. In our analyzed study, out of the total 14 intrauterine deaths, three were attributed to HELLP syndrome, three to AFLP, and one to hepatitis. Perinatal mortality was 16% in our study. Konareddy and Krithika[130] mentioned 25% incidence

of perinatal mortality in their study .Perinatal mortality was found to be 30.7% in the Mitta [129]study and 35.4 % in Umang Rathi et al study. [67] The lower incidence of maternal and fetal morbidity and mortality in our study appears to be due to more number of patients from urban population-more awareness, high female literacy rate, adequate medical facilities, increased maternal and child health care services and increased awareness regarding pregnancy specific conditions.

Liver disease during pregnancy often goes undetected and undiagnosed because LFTs are not typically part of routine antenatal tests. Additionally, the symptoms of hepatic dysfunction can be subtle and may resemble other pregnancy-specific conditions, making it difficult to identify liver issues.

Increasing awareness about liver diseases among pregnant women and health care providers can lead to earlier detection and intervention. Public health campaigns, educational programs and accessible resources can help spread knowledge about the symptoms and risks associated with liver conditions during pregnancy.

Managing pregnant women with jaundice is crucial because it affects both the mother and the unborn baby. The key to successful management lies in identifying the underlying cause of the jaundice early. By prioritizing early diagnosis and timely delivery, the materno-fetal risks of mortality and morbidity can be greatly minimized.

SUMMARY AND CONCLUSION

SUMMARY

- The study was conducted in 100 pregnant females with liver disease and 50 control pregnant females without liver disease over a 15 month period in a tertiary care center.
- LFTs were studied and materno-fetal outcome was compared between the two groups.
- There were 280 cases of liver disorder during pregnancy out of 1480 deliveries during a 15 month period out of which 100 cases fulfilling the criteria were taken into study. The incidence of liver disorders during pregnancy was found to be 18 % in our study.
- Out of 100 patients, 62 had pregnancy specific liver disorders,22 were found to have liver disorders unrelated to pregnancy and in 16 patients no definitive cause was found for the abnormal LFTs.
- ICP was the most prevalent liver disorder among pregnancy specific liver disorder accounting for 45 cases out of 62 (72%), followed by HELLP syndrome 6 cases out of 62 (9.6%).
- Tropical fever was the most common among liver disorders unrelated to pregnancy accounting for 11 cases out of 22 (50%), followed by Hepatitis 5 out of 22 (22.7%).
- In our study, the highest number of patients was within the 26-30 years age group (36%).
- Out of 100 patients in study group, 59 patients (59%) were multigravida while remaining 41 patients (41%) were primigravida. Among the control group, 35 patients (70%) were multigravida and remaining 15 (30%) were primigravida.
- Out of 100 patients in study group, maximum number of patients when presented 45 (45%) had gestational age > 36 weeks and In the control group out of 50 patients, 42 patients (84%) had gestational age > 36 weeks.
- Mean gestational age was 32 weeks in study group and 36 weeks in control group.

- In this study out of 100, the majority of patients 80 (80%) presented in the third trimester of pregnancy, with 49 presenting at term.16 patients (16%) presented in their second trimester, and 4 patients (4%) presented during the first trimester.
- Pain abdomen was the most observed symptom (49%) followed by yellowish discoloration (14%) and pruritus (12%).
- The most common sign seen was icterus (19%), followed by pallor (15%).
- Mean AST was 274.00, Mean ALT was 247.1 and Mean ALP was 262.51 in study group.
- Mean AST was 33.80, Mean ALT was 31.90 and Mean ALP was 151.12 in control group.
- Mean Total Bilirubin was 2.39, mean Direct Bilirubin was 1.73 in Study group. Mean Total Bilirubin was 0.46 and mean Direct Bilirubin was 0.21 in control group.
- C -section was most common mode of delivery in our study (78%) while in control group majority of patients had vaginal delivery (70%).
- The most common indication for C -section was fetal distress (36%) followed by scar tenderness (10%).
- Mean fetal birth weight was 2.2 kg in study group and 2.8 kg in control group.
- Among fetal outcome, Out of 100,48 babies had normal live birth (48%),22 had pre term birth (22%),14 had intrauterine death (14%),12 had fetal growth restriction (12%) and 4 abortions were there (4%).While In Control group, 47 babies had normal live birth (94%), 1 had pre term birth (2%) ,1 had intrauterine death (2%) and 1 still birth was there (2%).
- In fetal complications-30 babies were born prematurely (30%), 6 required NICU admission (6%) and 2 had perinatal asphyxia (2%).While In Control group, there were no complications observed.
- Regarding maternal outcome in study group-57 females had normal outcome (57%),40 had prolonged hospital stay (40%),2 had

multiorgan failure (2%) and 1 had maternal mortality (1%).In control group, 49 females had normal outcome (98%) and only 1 had prolonged hospital stay (2%).

- In maternal complications,8 patients had sepsis (8%) out of which 3 patients (3%) had septic shock,6 patients had HELLP (6%).5 patients had pre-eclampsia (5%),5 patients had acute liver failure (5%),4 patients had anemia (4%),3 patients had APH (3%),3 patients had obstetrical hysterectomy (3%),2 patients had acute kidney injury (2%), 1 patient had DIC (1%) and 1 patient developed pulmonary edema.

CONCLUSION

ICP was the most common pregnancy specific liver disorder in our study.Tropical fever was the most common among liver disorders unrelated to pregnancy in our study. Among study group 48% babies and 57% females had normal outcome while in control group 94% babies and 98% females had normal outcome. Therefore managing pregnant women with jaundice is crucial because it affects both the mother and the unborn baby. The key to successful management lies in identifying the underlying cause of the jaundice early. By prioritizing early diagnosis and timely delivery, the materno-fetal risks of mortality and morbidity can be greatly minimized.

BIBILOGRAPHY

[1] Ch'Ng CL, Morgan M, Hainsworth I, Kingham JG. Prospective study of liver dysfunction in pregnancy in Southwest Wales. Gut. 2002;51:876-80.

[2] Joshi D, James A, Quaglia A, Westbrook RH, Heneghan MA Ellington SR, Flowers L,et al. Liver disease in pregnancy. The Lancet. 2010;375:594-05.

[3] AP. Recent trends in hepatic diseases during pregnancy in the United States, 2002–2010. American journal of obstetrics and gynecology. 2015;21:524-1.

[4] Allen AM, Kim WR, Larson JJ, Rosedahl JK, Yawn BP, McKeon K, et al. The epidemiology of liver diseases unique to pregnancy in a US community: a population-based study. Clinical Gastroenterology and Hepatology. 2016;1:287-94.

[5] Westbrook RH, Dusheiko G, Williamson C. Pregnancy and liver disease. Journal of hepatology. 2014; 64:933-45.

[6] Mikolasevic I, Filipec-Kanizaj T, Jakopcic I, Majurec I, Brncic-Fischer A, Sobocan N,et al. Liver disease during pregnancy: a challenging clinical issue. Medical science monitor: international medical journal of experimental and clinical research. 2018;24:4080.

[7] Sanghavi M, Rutherford J.D. "Cardiovascular physiology of pregnancy."*Circulation.* 2014;130:1003-8

[8] Ushida T, Kotani T, Kinoshita F, Imai K, Nakano-Kobayashi T, Nakamura N, et al. Liver transaminase levels during pregnancy: a Japanese multicenter study. The Journal of Maternal-Fetal & Neonatal Medicine. 2022; 35:5761-67.

[9] Girling JC, Dow E, Smith JH. Liver function tests in pre-eclampsia: importance of comparison with a reference range derived for normal pregnancy. BJOG: An International Journal of Obstetrics & Gynaecology. 1997;104:246-50.

[10] Mitchell AL, Ovadia C, Syngelaki A, Souretis K, Martineau M, Girling J,et al. Re-evaluating diagnostic thresholds for intrahepatic cholestasis of pregnancy: case–control and cohort study. BJOG: An International Journal of Obstetrics & Gynaecology. 2021;128:1635-44.

[11] Goel A, Jamwal KD, Ramachandran A, Balasubramanian KA, Eapen CE. Pregnancy-related liver disorders. Journal of clinical and experimental hepatology. 2014;4:151-62.

[12] Kelly C, Pericleous M. Pregnancy-associated liver disease: a curriculum-based review. Frontline gastroenterology. 2018;9:170-4.

[13] Gomes CF, Sousa M, Lourenço I, Martins D, Torres J. Gastrointestinal diseases during pregnancy: what does the gastroenterologist need to know?. Annals of gastroenterology. 2018; 31:385.

[14] A.V. Sharma, S. John. “Pregnancy, liver disease”. StatPearls [Internet].StatPearls Publishing, (2018),

[15] Kamimura K, Abe H, Kawai H, Kamimura H, Kobayashi Y, Nomoto M, et al . Advances in understanding and treating liver diseases during pregnancy: A review. World Journal of Gastroenterology: WJG. 2015; 21:5183.

[16] Mishra N, Mishra VN, Thakur P. Study of abnormal liver function test during pregnancy in a tertiary care hospital in Chhattisgarh. The Journal of Obstetrics and Gynecology of India. 2016; 66:129-35.

[17] MKnight M, Nelson-Piercy C, Kurinczuk JJ, Spark P, Brocklehurst P. A prospective national study of acute fatty liver of pregnancy in the UK. Gut. 2008; 57:951-56.

[18] Nelson DB, Yost NP, Cunningham FG. Acute fatty liver of pregnancy: clinical outcomes and expected duration of recovery. American journal of obstetrics and gynecology. 2013; 209:456-61.

[19] Mikolasevic I, Filipec-Kanizaj T, Jakopcic I, Majurec I, Brncic-Fischer A, Sobocan N, et al. Liver disease during pregnancy: a challenging clinical issue. Medical science monitor: international medical journal of experimental and clinical research. 2018; 24:4080.

[20]. Murali AR, Devarbhavi H, Venkatachala PR, Singh R, Sheth KA. Factors that predict 1-month mortality in patients with pregnancy-specific liver disease. Clinical Gastroenterology and Hepatology. 2014; 12:109-13.

[21] Joseph KS, Boutin A, Lisonkova S, Muraca GM, Razaz N, John S, et al. Maternal mortality in the United States: recent trends, current status, and future considerations. Obstetrics & Gynecology. 2021;137:763-71.

[22] Ellington S.R.,Flowers L., Legardy-Williams J.K. et al. “Recent trends in hepatic diseases during pregnancy in the United States, 2002–2010.” Am J Obstet Gynecol. 2015; 212:524.27

[23] Sarkar M., Brady C.W., Fleckenstein J. et al.Reproductive health and liver disease: practice guidance by the American Association for the Study of Liver Diseases.Hepatology. 2021; 73: 318-65

[24] Shekhar S, Diddi G. Liver disease in pregnancy. Taiwan J Obstet Gynecol. 2015; 54:475-82.

[25] Italian Association for the Study of the Liver (AISF).AISF position paper on liver disease and pregnancy.Dig Liver Dis, 2016; 48:120-37

[26] Liu J, Ghaziani TT, Wolf JL. Acute fatty liver disease of pregnancy: updates in pathogenesis, diagnosis, and management. Official journal of the American College of Gastroenterology| ACG. 2017;112:838-46.

[27] Fell DB, Dodds L, Joseph KS, Allen VM, Butler B. Risk factors for hyperemesis gravidarum requiring hospital admission during pregnancy. Obstetrics & Gynecology. 2006;107:277-84.

[28] Fiaschi L, Nelson-Piercy C, Gibson J, Szatkowski L, Tata LJ. Adverse maternal and birth outcomes in women admitted to hospital for hyperemesis gravidarum: a population-based cohort study. Paediatric and perinatal epidemiology. 2018; 32:40-51.

[29] Walch A, Duke M., Auty T.Profound hypokalaemia resulting in maternal cardiac arrest: a catastrophic complication of hyperemesis gravidarum?.Case Rep Obstet Gynecol.2018:4687587.

[30] Florentin M., Parthymos I., Agouridis A.P.Hyperemesis gravidarum: a benign condition of pregnancy or a challenging metabolic disorder?.Eur J Case Rep Intern Med. 2020; 7001979.

[31] Munch S, Korst L.M., Hernandez G.D. Health-related quality of life in women with nausea and vomiting of pregnancy: the importance of psychosocial context.J Perinatol. 2011; 31: 10-20.

[32] Tan A, Lowe S., Henry A.Nausea and vomiting of pregnancy: Effects on quality of life and day-to-day function.Aust N Z J ObstetGynaecol. 2018; 58: 278-90.

[33] Nana M, Tydeman F., Bevan G. Hyperemesis gravidarum is associated with increased rates of termination of pregnancy and suicidal ideation: results from a survey completed by >5000 participants.Am J Obstet Gynecol. 2021; 224: 629-31.

[34] Poursharif B., Korst L.M., Macgibbon K.W. Elective pregnancy termination in a large cohort of women with hyperemesis gravidarum.Contraception. 2007; 76: 451-55.

[35] Fiaschi L., Nelson-Piercy C., Gibson J. Adverse maternal and birth outcomes in women admitted to hospital for hyperemesis gravidarum: a population-based cohort study.Paediatr Perinat Epidemiol. 2018; 32: 40-51.

[36] Varela P., Deltsidou A.Hyperemesis gravidarum and neonatal outcomes: a systematic review of observational studies.Taiwan J Obstet Gynecol. 2021; 60: 422-32.

[37] Fejzo M., Kam A., Laguna A. MacGibbon K, Mullin P. Analysis of neurodevelopmental delay in children exposed in utero to hyperemesis gravidarum reveals increased reporting of autism spectrum disorder. ReprodToxicol. 2019; 84: 59-64.

[38] Fejzo M.S. ,Magtira A. ,Schoenberg F.P. Neurodevelopmental delay in children exposed in utero to hyperemesis gravidarum.Eur J Obstet GynecolReprod Biol. 2015; 189: 79-84.

[39] Whitehouse A.J.O. ,Alvares G.A. ,Cleary D Symptom severity in autism spectrum disorder is related to the frequency and severity of nausea and vomiting during pregnancy: a retrospective case-control study.Mol Autism. 2018; 9: 1-8.

[40] Committee on Practice Bulletins-ObstetricsACOG Practice bulletin no. 189: nausea and vomiting of pregnancy.Obstet Gynecol. 2018; 131:15-30.

[41] Nelson-Piercy C, Dean C, Shehmar M, Gadsby R, O'Hara M, Hodson K, et al. Royal College of Obstetricians and Gynaecologists. The Management of Nausea and Vomiting in Pregnancy and Hyperemesis Gravidarum (Green-top Guideline No. 69). BJOG: An International Journal of Obstetrics & Gynaecology. 2024; 131:1-30.

[42] Lowe S.A., Armstrong G., Beech A. SOMANZ position paper on the management of nausea and vomiting in pregnancy and hyperemesis gravidarum. Aust N Z J ObstetGynaecol. 2020; 60: 34-43.

[43] Kaplan Y.C. ,Richardson J.L. ,Keskin-Arslan E. Use of ondansetron during pregnancy and the risk of major congenital malformations: a systematic review and meta-analysis. ReprodToxicol. 2019; 86: 1-13.

[44] ChiossiG. ,Neri I. ,Cavazzuti M. Hyperemesis gravidarum complicated by Wernicke encephalopathy: background, case report, and review of the literature. ObstetGynecolSurv. 2006; 61: 255-68.

[45] GrootenI.J. ,Koot M.H. ,van der Post J.A. Early enteral tube feeding in optimizing treatment of hyperemesis gravidarum: the Maternal and Offspring outcomes after Treatment of HyperEmesis by Refeeding (MOTHER) randomized controlled trial.Am J Clin Nutr. 2017; 106: 812-20.

[46] Turunen K, Helander K, Mattila KJ, Sumanen M. Intrahepatic cholestasis of pregnancy is common among patients' first-degree relatives. Acta ObstetGynecol Scand. 2013; 92:1108-110.

[47] Pataia V, Dixon PH, Williamson C. Pregnancy and bile acid disorders. Am J PhysiolGastrointest Liver Physiol. 2017; 313:1-6.

[48] Davit-Spraul A, Gonzales E, Baussan C, Jacquemin E. The spectrum of liver diseases related to ABCB4 gene mutations: pathophysiology and clinical aspects. Semin Liver Dis. 2010; 30:134-46.

[49] Anzivino C, Odoardi MR, Meschiari E, Baldelli E, Facchinetti F, Neri I, Ruggiero G,et al. ABCB4 and ABCB11 mutations in intrahepatic cholestasis of pregnancy in an Italian population. Dig Liver Dis. 2013; 45:226-32.

[50] Dixon PH, Williamson C. The pathophysiology of intrahepatic cholestasis of pregnancy. Clin Res Hepatol Gastroenterol. 2016; 40:141-53.

[51] Keitel V, Vogt C, Häussinger D, Kubitz R. Combined mutations of canalicular transporter proteins cause severe intrahepatic cholestasis of pregnancy. Gastroenterology. 2006; 131:624-29.

[52] Sookoian S, Castaño G, Burgueño A, Gianotti TF, Pirola CJ. Association of the multidrug-resistance-associated protein gene

(ABCC2) variants with intrahepatic cholestasis of pregnancy. J Hepatol. 2008; 48:125-32.

[53] Van Mil SW, Milona A, Dixon PH, Mullenbach R, Geenes VL, Chambers J,et al. Functional variants of the central bile acid sensor FXR identified in intrahepatic cholestasis of pregnancy. Gastroenterology. 2007;133:507-16.

[54] Bull LN, Thompson RJ. Progressive Familial Intrahepatic Cholestasis. Clin Liver Dis. 2018; 22:657-69.

[55] Mutlu MF, Aslan K, Guler I, Mutlu I, Erdem M, Bozkurt N, et al. Two cases of first onset intrahepatic cholestasis of pregnancy associated with moderate ovarian hyperstimulation syndrome after IVF treatment and review of the literature. J ObstetGynaecol. 2017; 37:547-49.

[56] Gabzdyl EM, Schlaeger JM. Intrahepatic cholestasis of pregnancy: a critical clinical review. J Perinat Neonatal Nurs. 2015;29:41-50.

[57] Abu-Hayyeh S, Papacleovoulou G, Lövgren-Sandblom A, Tahir M, Oduwole O, Jamaludin NA, et al. 'Intrahepatic cholestasis of pregnancy levels of sulfated progesterone metabolites inhibit farnesoid X receptor resulting in a cholestatic phenotype', *Hepatology* 2013;57:716-26.

[58] Geenes V, Williamson C. Intrahepatic cholestasis of pregnancy. World J Gastroenterol. 2009; 15:2049-66.

[59] Floreani A, Gervasi MT. New Insights on Intrahepatic Cholestasis of Pregnancy. Clin Liver Dis. 2016; 20:177-89.

[60] Marschall HU, Wikström Shemer E, Ludvigsson JF, Stephansson O. Intrahepatic cholestasis of pregnancy and associated hepatobiliary disease: a population-based cohort study. Hepatology. 2013; 58:1385-391

[61] Kondrackiene J, Kupcinskas L. Intrahepatic cholestasis of pregnancy-current achievements and unsolved problems. World J Gastroenterol. 2008; 14:5781-88.

[62]. Manzotti C, Casazza G, Stimac T, Nikolova D, Gluud C. Total serum bile acids or serum bile acid profile, or both, for the diagnosis of intrahepatic cholestasis of pregnancy. Cochrane Database Syst Rev. 2019;7:12546.

[63]. Lee NM, Brady CW. Liver disease in pregnancy. World J Gastroenterol. 2009; 15:897-906.

[64]. Maldonado M, Alhousseini A, Awadalla M, Idler J, Welch R, Puder K, et al. Intrahepatic Cholestasis of Pregnancy Leading to Severe Vitamin K Deficiency and Coagulopathy. Case Rep Obstet Gynecol. 2017; 2017:5646247.

[65]. Eapen C.E., Ramakrishna B., Jose R., Loganathan G., Chandy G. Liver failure during pregnancy. *Gut.* 2008; 57:83.

[66]. Knight M, Nelson-Piercy C., Kurinczuk J.J., Spark P., Brocklehurst P. A prospective national study of acute fatty liver of pregnancy in the UK. *Gut.* 2008;57:951–56.

[67] Rathi U, Bapat M, Rathi P, Abraham P. Effect of liver disease on maternal and fetal outcome--a prospective study. Indian J Gastroenterol. 2007; 26:59–63.

[68] Natarajan SK, Thangaraj KR, Goel A, Eapen CE, Balasubramanian KA, Ramachandran A et al. Acute fatty liver of pregnancy: an update on mechanisms. Obstet Med: 2011;4:99-103.

[69]. Natarajan S.K., Thangaraj K.R., Eapen C.E. Liver injury in acute fatty liver of pregnancy: possible link to placental mitochondrial dysfunction and oxidative stress. *Hepatology.* 2010;51:191-200.

[70] Ibdah J.A., Bennett M.J., Rinaldo P. A fetal fatty-acid oxidation disorder as a cause of liver disease in pregnant women. *N Engl J Med.* 1999;340:1723-31.

[71] Yang Z., Yamada J., Zhao Y., Strauss A.W., Ibdah J.A. Prospective screening for pediatric mitochondrial trifunctional protein defects in pregnancies complicated by liver disease. *Jama.* 2002; 288:2163-66.

[72] Innes A.M., Seargeant L.E., Balachandra K. Hepatic carnitine palmitoyltransferase I deficiency presenting as maternal illness in pregnancy. *Pediatr Res.* 2000; 47:43-45.

[73] Browning M.F., Levy H.L., Wilkins-Haug L.E., Larson C., Shih V.E. Fetal fatty acid oxidation defects and maternal liver disease in pregnancy. *Obstet Gynecol.* 2006; 107:115-20.

[74] Maitra A., Domiati-Saad R., Yost N., Cunningham G., Rogers B.B., Bennett M.J. Absence of the G1528C (E474Q) mutation in the alpha-subunit of the mitochondrial trifunctional protein in women with acute fatty liver of pregnancy. *Pediatr Res.* 2002;51:658-61.

[75] Kong X.F., Zhang X.X., Yu Y.Y. No mutation was found in the alpha-subunit of the mitochondrial tri-functional protein in one patient with severe acute fatty liver of pregnancy and her relatives. *J Gastroenterol Hepatol.* 2007; 22:2107–111.

[76] Steegers EA, von Dadelszen P, Duvekot JJ, Pijnenborg R. Pre-eclampsia.*Lancet.* 2010; 376: 631-44.

[77] Khan KS, Wojdyla D, Say L, Gülmezoglu AM, Van Look PF. WHO analysis of causes of maternal death: a systematic review.*Lancet.* 2006; 367: 1066-74.

[78] Souza J.P ,Gulmezoglu A.M,Vogel J. Moving beyond essential interventions for reduction of maternal mortality (the WHO Multicountry Survey on Maternal and Newborn Health): a cross-sectional study.*Lancet.* 2013; 381: 1747-755.

[79] Wallis AB, Saftlas AF, Hsia J, Atrash HK. Secular trends in the rates of preeclampsia, eclampsia, and gestational hypertension, United States, 1987–2004.*Am J Hypertens.* 2008; 21: 521-26.

[80] Ananth CV, Keyes KM, Wapner RJ. Pre-eclampsia rates in the United States, 1980-2010: age-period-cohort analysis. BMJ. 2013;347:6564.

[81] Trottmann F, Baumann M, Amylidi-Mohr S, Surbek D, Risch L, Mosimann B, et al. Angiogenic profiling in HELLP syndrome cases with or without hypertension and proteinuria.*Eur J Obstet Gynecol Reprod Biol.* 2019; 243: 93-96.

[82] Deruelle P, Coudoux E, Ego A, Houfflin-Debarge V, Codaccioni X, Subtil D. Risk factors for post-partum complications occurring after preeclampsia and HELLP syndrome. A study in 453 consecutive pregnancies.*Eur J ObstetGynecolReprod Biol.* 2006; 125: 59-65.

[83] Weinstein L.Syndrome of hemolysis, elevated liver enzymes, and low platelet count: a severe consequence of hypertension in pregnancy.*Am J Obstet Gynecol.* 1982; 142: 159-67.

[84] Sibai BM, Ramadan MK, Usta I, Salama M, Mercer BM, Friedman SA. Maternal morbidity and mortality in 442 pregnancies with hemolysis, elevated liver enzymes, and low platelets (HELLP syndrome).*Am J Obstet Gynecol.* 1993; 169: 1000-6.

[85] Lisonkova S, Bone JN, Muraca GM, Razaz N, Wang LQ, Sabr Y, et al. Incidence and risk factors for severe preeclampsia, HELLP syndrome and eclampsia at preterm and term gestation: a population-based study.*Am J Obstet Gynecol.* 2021; 225: 538.1-19.

[86] Malmstrom O. Morken N.H. HELLP syndrome, risk factors in first and second pregnancy: a population-based cohort study. *Acta Obstet Gynecol Scand.* 2018; 97: 709-16.

[87] Gomez-Tolub R, Rabinovich A, Kachko E, Benshalom-Tirosh N, Tirosh D, Thachil J et al. Placental abruption as a trigger of DIC in women with HELLP syndrome: a population-based study [published online ahead of print Septemb, 2020. J Matern Fetal Neonatal Med 2022 ;35:3259-69.

[88] Gasem T. Al Jama F. E. Burshaid S. Rahman J, Al Suleiman SA, Rahman MS. Maternal and fetal outcome of pregnancy complicated by HELLP syndrome.*J Matern Fetal Neonatal Med.* 2009; 22: 1140-43.

[89] Tolunay HE, Kahraman NC, Varli EN, Reis YA, Celen S, Caglar AT. Can first-trimester AST to platelet ratio index scores predict HELLP syndrome?.*J Coll Physicians Surg Pak.* 2021; 31: 188-92.

[90] Gedik E, Yücel N, Sahin T, Koca E, Colak YZ, Togal T. Hemolysis, elevated liver enzymes, and low platelet syndrome: Outcomes for patients admitted to intensive care at a tertiary referral hospital. Hypertension in pregnancy. 2017;36:21-9.

[91] Erdemoğlu M, Kuyumcuoğlu U, Kale A, Akdeniz N. Factors affecting maternal and perinatal outcomes in HELLP syndrome: evaluation of 126 cases. Clin Exp Obstet Gynecol. 2010;37:213-6.

[92] Carpani G. Bozzo M. Ferrazzi E, D'Amato B, Pizzotti D, Radaelli T, et al. The evaluation of maternal parameters at diagnosis may predict HELLP syndrome severity. *J Matern Fetal Neonatal Med.* 2003; 13: 147-51.

[93] Chou MM, Chen YF, Kung HF, Liu CK, Sun L, Chen WC, et al.Extensive hepatic infarction in severe preeclampsia as part of the HELLP syndrome (hemolysis, elevated liver enzymes, and low platelets): evolution of CT findings and successful treatment with plasma exchange therapy.*Taiwan J Obstet Gynecol.* 2012; 51: 418-20.

[94] Devarbhavi H, Kremers WK, Dierkhising R, Padmanabhan L. Pregnancy-associated acute liver disease and acute viral hepatitis: differentiation, course and outcome.*J Hepatol.* 2008; 49: 930-5.

[95] Devarbhavi H, Rao P, Patil M, Adarsh CK. Characteristics of ascites in patients with pregnancy-specific liver diseases.*Clin Gastroenterol Hepatol.* 2012; 10: 559-62.

[96] Vigil-De Gracia P. Acute fatty liver and HELLP syndrome: two distinct pregnancy disorders. *Int J Gynaecol Obstet.* 2001; 73: 215-20.

[97] Ang SX, Chen CP, Sun FJ, Chen CY. Comparison of maternal and neonatal outcomes between acute fatty liver of pregnancy and hemolysis, elevated liver enzymes and low platelets syndrome: a retrospective cohort study.*BMC Pregnancy Childbirth.* 2021; 21: 293.

[98] MBRRACE-UK. Saving Lives, Improving Mothers' Care. 2021. Available at: https://www.npeu.ox.ac.uk/assets/downloads/mbrrace-uk/reports/maternalreport2021/MBRRACEUK_Maternal_Report_2021_-_FINAL_-_WEB_VERSION.pdf.Accessed October 30, 2021.

[99] Yücesoy G, Özkan S, Bodur H, Tan T, Çalışkan E, Vural B, et al. Maternal and perinatal outcome in pregnancies complicated with hypertensive disorder of pregnancy: a seven year experience of a tertiary care center.*Arch Gynecol Obstet.* 2005; 273: 43-9.

[100] Chhabra S. Kakani A. Maternal mortality due to eclamptic and non-eclamptic hypertensive disorders: a challenge. *J ObstetGynaecol.* 2007; 27: 25-29.

[101] Lisonkova S, Razaz N, Sabr Y, Muraca GM, Boutin A, Mayer C, et al. Maternal risk factors and adverse birth outcomes associated with HELLP syndrome: a population-based study.*BJOG.* 2020;127:1189-98.

[102] Nunes JO, Turner MA, Fulcher AS. Abdominal imaging features of HELLP syndrome: a 10-year retrospective review.*AJR Am J Roentgenol.* 2005;185: 1205-10.

[103] Dubey S. Rani J. Hepatic rupture in preeclampsia and HELLP syndrome: A catastrophic presentation. *Taiwan J Obstet Gynecol.* 2020; 59: 643-51.

[104] Henny CP, Lim TE, Brummelkamp WH, Büller HR, ten Cate JW. Spontaneous rupture of Glisson's capsule during pregnancy. An acute surgical emergency. The Netherlands journal of surgery. 1982;34:72-5.

[105] Grand'Maison S, Sauvé N, Weber F, Dagenais M, Durand M, Mahone M. Hepatic rupture in hemolysis, elevated liver enzymes, low platelets syndrome.*Obstet Gynecol.* 2012;119:617-25.

[106] Pereira SP, O'Donohue J, Wendon J, Williams R. Maternal and perinatal outcome in severe pregnancy-related liver disease. Hepatology. 1997 Nov 1;26(5):1258-62.

[107] García-Romero CS, Guzman C, Cervantes A, Cerbón M. Liver disease in pregnancy: Medical aspects and their implications for mother and child. Annals of hepatology. 2019;18:553-62.

[108] Mahajan A. Gupta ‘Study of liver diseases in pregnancy & the maternal outcomes’ International Journal of Life Sciences, Biotechnology and Pharma Research 2023;12.

[109] Audibert F, Friedman SA, Frangieh AY, Sibai BM. Clinical utility of strict diagnostic criteria for the HELLP (hemolysis, elevated liver enzymes, and low platelets) syndrome. American journal of obstetrics and gynecology. 1996;175:460-4.

[110] Satia MN, Jandhyala M. A study of fetomaternal outcomes in cases of jaundice at a tertiary care centre. Int J Reprod Contracept Obstet Gynecol. 2016;5:2352-7.

[111] Sharma S, Aherwar R, Jawade S. Maternal and fetal outcome in jaundice complicating pregnancy: a prospective study. Int J Reprod Contracept Obstet Gynecol 2016;5:1084-87.

[112] Jain M, Thaker H. A study of fetomaternal outcome of hepatic disorders in pregnancy. Int J Reprod Contracept Obstet Gynecol 2019;8:1182-85.

[113] Tiwari A, Aditya V, Srivastava R, Gupta G. A study of spectrum and outcome of liver diseases in pregnant women at BRD medical college. Int J Reprod Contracept ObstetGynecol2017; 6:3641-5.

[114] Patra KK, Chattopadhyay S, Biswas S, Hadi MA. A study of abnormal liver function in pregnancy and its correlation with fetomaternal outcome in a teaching hospital of Kolkata in Eastern India. Asian Journal of Medical Sciences. 2022;13:80-6.

[115] Sarkar CS, Giri AK, Maity TK. Jaundice in pregnancy: a clinical study. J Indian Med Assoc. 1992;90:117-8.

[116] [neema]Acharya N, Acharya S, Shukla S, et al. Study of jaundice in pregnancy. Global J Med Res 2014. 13.

[117] Krishnamoorthy J, Murugesan A. Jaundice during pregnancy: maternal and fetal outcome. Int J Reproduct, Contracept, ObstetGynecol 2017;5:2541-6.

[118] Vinayachandran SN, Anaswara K. Liver disorders in pregnancy: a fetomaternal outcome. J South Asian Feder Obst Gynaecol. 2020;12:167-71.

[119] Bacq Y, Sapey T, Brechot M, Pierre F, Fignon A, Dubois F. Intrahepatic cholestasis of pregnancy: a French prospective study. Hepatology. 1997;26:358-64.

[120] Dhawan PS, Sainani GS. Diagnosis of liver disorder in pregnancy. Am J Obstetrics and Gynaecology. 1996; 23:609-13.

[121] Panchbudhe SA, Parulekar M, Kudale S. Liver Disease in Pregnancy: A Prospective Analysis of Maternal and Fetal Outcomes among 120 Patients at a Tertiary Care Center. Journal of South Asian Federation of Obstetrics and Gynaecology. 2022;14:551-6.

[122] Tank PD, Nadanwar YS and Mayadeo NM. Outcome of pregnancy with severe liver disease. Int J Gynecol Obstet. 2002;76:27-31.

[123] Shukla S, Mehta G, Jais M, Singh A. A prospective study on acute viral hepatitis in pregnancy; seroprevalence, and fetomaternal outcome of 100 cases. J biosci Tech 2011; 2:279-86.

[124] Devarbhavi H, Kremers WK, Dierkhising R, Padmanabhan L. Pregnancy-associated acute liver disease and acute viral hepatitis: differentiation, course and outcome. Journal of hepatology. 2008;49:930-5.

[125] Suri AD, Jain RK, Jain SC. Study of Jaundice profile in Pregnancy in tertiary care centre in central India. Int J Med Res Rev. 2014;2:3-7.

[126] Kohli UA, Seth A, Singh S, Mishra R. Liver ailments in pregnancy: our experience Int J Reprod Contracept ObstetGynecol 2017;6:939-43.

[127] Tripti N, Sarita A. Fetomaternal outcome in jaundice during pregnancy. ObstetGynecol of India. 2005;55:424-27.

[128] Nagaria T, Agarwal S. Fetomaternal outcome in jaundice during pregnancy. J ObstetGynecol India 2005; 55:424-27.

[129] Mitta P, Rao SV. Fetomaternal outcome in jaundice complicating pregnancy. IOSR J Dent Med Sci 2016;15:72-76.

[130] Konareddy T, Krithika KA. Jaundice in pregnancy, a clinical study at JSS hospital, Mysore, Karnataka, India. Int J Reprod Contracept Obstet Gynaecol.2016; 5:2257-60.

KEYS TO MASTER CHART

ACLF	Acute on chronic liver failure
APH	Antepartum haemorrhage
ALP	Alkaline phosphatase
ALT	Alanine aminotransferase
AST	Aspartate aminotransferase
BP	Blood pressure
DIC	Disseminated intravascular Coagulation
EDD	End date of delivery
HAV	Hepatitis A virus
Hb	Haemoglobin
HBsAg	Hepatitis B surface antigen
HCV	Hepatitis C virus
HEV	Hepatitis E virus
HIV	Human immunodeficiency virus
HELLP	Haemolysis, elevated liver enzymes, low platelet count
ICP	Intrahepatic cholestasis of Pregnancy
LSCS	Lower segment caesarean Section
LMP	Last menstrual period
NA	Not applicable
NR	Non reactive
NICU	Neonatal intensive unit

SR.No.	Name	MRD No.	GROUP	Age (years)	Gravida	Trimester	LMP	EDD	Gestation Age (weeks)	Co Morbidities	Fever	Persistent Nausea/ Vomiting	Yellowish Discoloration	Pain Abdomen	Itching	Difficulty in Breathing	Decreased Fetal Movements
1	PB	1462387	CASES	32	Primigravida	3rd	04-06-2022	03-11-2023	37	No	No	No	No	Yes	No	No	No
2	S	1413782	CASES	25	Primigravida	3rd	11-03-2022	16-12-2022	35	No	No	No	No	Yes	No	No	No
3	B	1402032	CASES	23	Primigravida	3rd	03-05-2022	12-10-2022	33	No	No	No	Yes	Yes	No	No	No
4	KD	1451526	CASES	31	Multigravida	3rd	26-04-2022	31-01-2023	39	No	No	No	No	Yes	No	No	No
5	JR	1448410	CASES	36	Primigravida	3rd	04-05-2022	11-02-2023	38	No	No	No	Yes	Yes	No	Yes	No
6	SJ	1443245	CASES	38	Multigravida	3rd	30-05-2022	10-02-2023	38	Yes	No	No	No	Yes	No	No	No
7	AT	1461767	CASES	31	Primigravida	3rd	21-05-2022	28-02-2023	38	No	No	No	No	Yes	No	No	No
8	IS	971104	CASES	33	Multigravida	3rd	27-05-2022	03-03-2023	33	No	No	No	No	Yes	No	No	No
9	S	426604	CASES	33	Primigravida	3rd	05-04-2022	02-11-2023	33	No	No	No	No	No	Yes	No	No
10	C	1451505	CASES	29	Primigravida	2nd	28-06-2022	04-04-2023	24	No	No	No	Yes	No	No	No	No
11	NB	1377020	CASES	21	Primigravida	3rd	26-05-2022	03-02-2023	37	No	No	No	No	Yes	No	No	No
12	SK	1330981	CASES	29	Multigravida	2nd	08-03-2022	05-10-2023	25	No	No	No	No	No	No	No	No
13	MR	1440358	CASES	24	Multigravida	3rd	04-06-2022	01-11-2023	37	No	No	Yes	Yes	Yes	No	Yes	No
14	IK	1274293	CASES	28	Primigravida	3rd	05-11-2022	18-02-2023	36	No	No	No	No	No	No	No	No
15	DV	439015	CASES	29	Multigravida	3rd	13-04-2022	20-02-2023	36	No	No	No	No	Yes	No	No	No
16	SM	1425249	CASES	41	Primigravida	3rd	21-03-2022	26-12-2022	36	No	No	No	No	No	No	No	Yes
17	SK	1485188	CASES	32	Multigravida	3rd	07-11-2022	18-04-2023	36	No	No	No	No	Yes	No	No	No
18	MD	1440877	CASES	32	Multigravida	3rd	20-06-2022	27-03-2023	32	No	No	No	Yes	Yes	No	No	No
19	HK	1441793	CASES	30	Primigravida	2nd	09-01-2022	06-08-2023	27	No	No	No	No	No	No	No	No
20	AK	1320450	CASES	32	Primigravida	1st	01-09-2022	06-08-2023	10	Yes	No	Yes	No	No	No	No	No
21	Lk	1517215	CASES	32	Multigravida	3rd	08-09-2022	15-06-2023	35	No	No	No	No	No	No	No	Yes
22	P	1062815	CASES	29	Multigravida	3rd	02-07-2022	09-04-2023	39	No	No	No	No	Yes	No	No	No
23	S	1513535	CASES	28	Multigravida	3rd	26-08-2022	02-06-2023	36	No	No	No	No	No	No	No	Yes
24	Sj	1514299	CASES	36	Primigravida	3rd	18-09-2022	25-06-2023	33	Yes	No	No	No	No	No	No	Yes
25	BK	1515638	CASES	37	Multigravida	3rd	12-10-2022	19-07-2023	30	No	No	No	No	Yes	No	No	No
26	S	1515601	CASES	27	Multigravida	3rd	06-08-2023	13-05-2023	35	No	No	No	No	Yes	No	No	No
27	K	1440202	CASES	31	Primigravida	3rd	15-05-2022	22-02-2023	32	Yes	No	No	No	No	No	No	No
28	MU	1390319	CASES	27	Primigravida	2nd	28-10-2022	04-08-2023	24	No	No	No	No	Yes	No	No	No
29	C	1490238	CASES	29	Multigravida	3rd	21-08-2022	28-05-2023	35	No	No	No	No	No	No	No	Yes
30	VK	1500715	CASES	30	Primigravida	3rd	02-08-2022	17-05-2023	35	No	No	No	No	No	No	No	No
31	A	1484650	CASES	21	Multigravida	2nd	05-11-2023	12-08-2023	19	No	No	No	Yes	No	Yes	No	No
32	SK	1474903	CASES	32	Primigravida	3rd	13-06-2022	20-03-2023	37	No	Yes	No	No	No	No	No	No
33	CN	1457193	CASES	28	Primigravida	3rd	20-05-2022	27-02-2023	37	No	No	No	No	Yes	No	No	No

SR.No.	Bleeding Per Vaginum/ Spotting	Pallor	Icterus	Hepatomegaly	Ascitis	Increased BP readings	ICP	Hyperemesis Gravidarum	Acute Liver Failure	HELLP	Acute Fatty Liver of Pregnancy	Pre eclampsia	Acute on Chronic Liver Failure	Drug Induced Liver Injury	Hb	TLC	Platelet count	AST	ALT
1	No	No	No	No	No	No	Mild								10.7	17.15	188	156	209
2	Yes	No	Yes	No	No	No				Yes					8.3	7.5	54	155	139
3	No	No	Yes	No	No	No									11.6	12.6	200	1390	1378
4	No	No	No	No	No	No									12.6	14.5	96	100	107
5	No	Yes	Yes	No	No	No				Yes					5.2	31.9	122	2743	1275
6	No	No	No	No	No	No									10.6	12.14	248	145	264
7	Yes	No	No	Yes	No	No	Moderate								13.1	20.3	233	259	317
8	No	No	No	No	No	No	Mild								10.6	9.3	164	86	146
9	No	No	No	No	No	No	Moderate				Yes				11	8.8	158	241	380
10	No	No	Yes	No	No	No	Severe								10.4	11.6	183	1948	921
11	No	No	No	No	No	No	Mild								10.8	8.86	271	63	101
12	No	No	No	No	No	No	Moderate								10	20.1	448	361	553
13	No	Yes	Yes	No	Yes	No									14.7	9	150	161	149
14	No	Yes	Yes	No	No	No									10.8	10.1	150	105	217
15	No	No	No	No	No	No	Moderate								10.5	10.4	220	99	126
16	No	No	No	No	No	Yes				Yes					9.4	8.3	170	175	114
17	No	No	No	No	No	No	Mild								13.6	11.5	120	299	216
18	No	No	No	No	No	No									9.5	8.06	362	271	330
19	Yes	No	No	No	No	No									9.9	8.8	253	114	210
20	No	No	Yes	No	No	No		Yes							9.3	18.5	331	75	201
21	NO	No	No	No	No	No	Mild								10.9	11.21	150	124	178
22	No	No	No	No	No	No									10.4	10.3	134	168	139
23	No	No	No	No	No	No									12.1	9.1	130	122	210
24	No	No	No	No	No	No	Moderate								11.7	13.2	156	580	614
25	Yes	No	No	No	No	No									9.78	13.7	322	165	138
26	No	Yes	No	No	No	No									5.4	22.5	100	104	75
27	No	No	No	No	No	Yes						Yes			12.6	13	177	63	108
28	No	No	No	No	No	No				Yes					10.7	18.6	231	236	375
29	No	Yes	No	No	No	No	Moderate								11.5	9.19	140	121	154
30	Yes	Yes	No	No	Yes	No									7.5	15.7	167	146	156
31	No	No	Yes	No	No	No									11.2	12.1	215	124	120
32	No	No	No	No	No	No	Mild								12.7	10.8	156	141	131
33	No	No	No	No	No	No	Moderate								11.1	9.9	158	107	146

SR.No.	ALP	Bile acids	Total Bilirubin	D. Bilirubin	Total Protein	S. Albumin	HIV	HAV	HEV	HBsAg	HCV	Dengue	Leptospirosis	Scrub typhus	Mode of delivery	Indications Of Cesarean Section	Fetal GENDER	Fetal Weight
1	265	24.3	0.9	0.4	7	3.24	NR	NA	NA	NR	NR	Negative	Negative	Negative	LSCS	Fetal distress	Female	3
2	197		17.5	14.7	5.4	2.74	NR	REACTIVE	NR	NR	NR	Negative	Negative	Negative	LSCS	Deteriorating Maternal condition	Female	2.4
3	253		11.89	10.5	5.9	3.05	NR	NR	REACTIVE	NR	NR	Negative	Negative	Negative	Preterm NVD		Female	2.1
4	276		0.68	0.14	7.2	3.53	NR	NR	NR	NR	NR	Negative	Negative	Negative	LSCS	Scar tenderness	Male	2.9
5	145		0.8	0.47	5.1	2.87	NR	NR	NR	NR	NR	Negative	Negative	Negative	Term NVD		Female	1.2
6	107		0.36	0.07	4.8	2.51	NR	NR	NR	NR	NR	Negative	Negative	Negative	LSCS	Scar tenderness	Male	2.76
7	447	56.8	1.61	1.57	5	2.7	NR	NR	NR	NR	NR	Negative	Negative	Negative	Term NVD		Female	2.76
8	183	29.8	0.56	0.09	6.2	3.3	NR	NA	NA	NR	NR	Negative	Negative	Negative	LSCS	Arrest of descent of head in 2nd stage of labour	Female	2.72
9	273	83.6	0.61	0.32	6.8	3.5	NR	NR	NR	NR	NR	Negative	Negative	Negative	LSCS	Moderate ICP	Male	2.8
10	239	184.4	12.18	10.33	5.6	3.04	NR	NR	REACTIVE	NR	NR	Negative	Negative	Positive	LSCS	Severe ICP	Female	1.36
11	295	35	0.15	0.12	6.6	3.26	NA	NA	NA	NR	NR	Negative	Negative	Negative	LSCS	Fetal distress	Male	2.4
12	258	85.7	0.55	0.14	7	4.12	NA	NA	NA	NR	NR	Negative	Negative	Negative	Preterm NVD		Female	1.08
13	653		9.44	7.65	5.8	2.55	NR	NR	NR	NR	NR	Negative	Negative	Negative	LSCS	Deteriorating Maternal condition	Male	0.8
14	382		0.64	0.22	7.1	3.14	NR	NR	NR	NR	NR	Negative	Negative	Negative	LSCS		Female	2.8
15	303	49.2	0.04	0.02	7.1	3.41	NR	NR	NR	NR	NR	Negative	Negative	Negative	LSCS	Previous LSCS	Female	2.5
16	232	11.7	0.21	0.07	7	3.92	NR	NR	NR	NR	NR	Negative	Negative	Negative	LSCS	Scar tenderness	Female	2.7
17	676	24.2	1.47	0.73	6.7	3.61	NR	NA	NA	NR	NR	Negative	Negative	Negative	LSCS	Scar tenderness	Female	2.4
18	278		4.24	2.17	7	2.82	NR	NR	REACTIVE	NR	NR	Negative	Negative	Negative	LSCS	Scar tenderness	Male	1.9
19	94	8.1	0.62	0.26	6.5	3.34	NR	NR	NR	NR	NR	Negative	Negative	Negative	LSCS	Fetal distress	Male	0.6
20	135		2.77	1.5	5.2	2.66	NR	NR	NR	NR	NR	Negative	Positive	Negative	Preterm NVD		Abortion	
21	62	22.9	2.17	1.56	3.6	2.14	NR	NR	NR	NR	NR	Negative	Negative	Negative	LSCS	Type IV placenta previa	Male	2.66
22	248		0.83	0.3	7.2	3.46	NR	NR	NR	NR	NR	Negative	Negative	Negative	Term NVD		Male	3.26
23	262	13.6	0.81	0.2	7.6	3.5	NR	NA	NA	NR	NR	Negative	Negative	Negative	LSCS	Fetal distress	Male	1.8
24	174	57.1	0.6	0.42	5.7	3.13	NR	NA	NA	NR	NR	Negative	Negative	Negative	LSCS	Fetal distress	Female	1.2
25	226		0.58	0.16	5.9	3.27	NR	NA	NA	NR	NR	Negative	Negative	Negative	LSCS	Previous LSCS	Male	1.8
26	1504		2.36	1.09	5.6	2.88	NR	NA	NA	NR	NR	Negative	Negative	Negative	Term NVD		Female	2.54
27	208		0.3	0.06	5.5	2.83	NR	NA	NA	NR	NR	Negative	Negative	Negative	LSCS	Fetal distress	Male	1.56
28	74	13.3	0.41	0.23	6.5	3.86	NR	NA	NA	NR	NR	Negative	Negative	Negative	Preterm NVD		Female	0.7
29	524	58.9	1.48	0.41	6.9	3	NR	NA	NA	NR	NR	Negative	Negative	Negative	LSCS	Fetal distress	Male Male	1.32,2.2
30	186		0.53	0.18	4.6	2.68	NR	NA	NA	NR	NR	Negative	Negative	Negative	LSCS	Atonic PPH	Female	2.6
31	220		3.77	3.28	5.9	2.71	NR	NR	NR	NR	NR	Negative	Negative	Negative	Preterm NVD		Abortion	0.32
32	367	25.1	2.99	1.65	6.5	3.09	NR	NA	NA	NR	NR	Negative	Negative	Negative	LSCS	Fetal distress	Female	3.2
33	370	51.4	0.43	0.27	6.9	3.38	NR	NR	NR	NR	NR	Negative	Negative	Negative	LSCS	Fetal distress	Female	3

SR.No.	Fetal Outcome	Fetal complications (If Any)	Maternal Outcome	Maternal Complications (If Any)	Disease
1	Normal Live Birth		Prolonged Hospital stay		Mild ICP
2	Fetal Growth restriction	NICU admission	Prolonged Hospital stay	HELLP	HELLP,Acute Viral Hepatitis
3	Preterm birth	Prematurity	Normal		Acute Viral Hepatitis
4	Normal Live Birth		Normal		
5	Intrauterine Death		Prolonged Hospital stay	Acute kidney injury,Pulmonary edema,HELLP	HELLP
6	Normal Live Birth		Normal		
7	Normal Live Birth		Normal		Moderate ICP
8	Preterm birth	Prematurity	Normal		Mild ICP
9	Preterm birth	Prematurity	Prolonged Hospital stay	Acute Liver Failure	Acute fatty liver of pregnancy,Acute Viral Hepatitis,Moderate ICP
10	Preterm birth	NICU admission	Prolonged Hospital stay		Acute Viral Hepatitis,Severe ICP
11	Normal Live Birth		Normal		Mild ICP
12	Intrauterine Death		Prolonged Hospital stay		Moderate ICP
13	Intrauterine Death		Multiorgan failure	DIC,Sepsis,Septic shock	
14	Normal Live Birth		Prolonged Hospital stay	Sepsis,Septic shock	
15	Normal Live Birth		Normal		Moderate ICP
16	Normal Live Birth		Normal	HELLP	HELLP
17	Normal Live Birth		Normal		Mild ICP
18	Intrauterine Death	Perinatal Asphyxia	Prolonged Hospital stay	Acute Liver Failure	Acute VIral Hepatitis
19	Intrauterine Death		Prolonged Hospital stay		
20	Abortion		Prolonged Hospital stay		Hyperemesis Gravidarum
21	Normal Live Birth		Prolonged Hospital stay	Obstetrical hysterectomy	Mild ICP
22	Normal Live Birth		Normal		
23	Fetal Growth restriction		Prolonged Hospital stay		
24	Fetal Growth restriction		Prolonged Hospital stay	Sepsis	Moderate ICP
25	Preterm birth	Prematurity	Normal		
26	Intrauterine Death		Normal	Sepsis,Anemia	
27	Preterm birth	Prematurity	Normal	Pre -eclampsia	Pre eclampsia
28	Intrauterine Death		Prolonged Hospital stay	Sepsis,HELLP	HELLP
29	Fetal Growth restriction		Prolonged Hospital stay		Moderate ICP
30	Normal Live Birth		Normal	Anemia	
31	Abortion		Normal		
32	Normal Live Birth		Normal		Mild ICP
33	Normal Live Birth		Normal		Moderate ICP

SR.No.	Name	MRD No.	GROUP	Age (years)	Gravida	Trimester	LMP	EDD	Gestation Age (weeks)	Co Morbidities	Fever	Persistent Nausea/ Vomiting	Yellowish Discoloration	Pain Abdomen	Itching	Difficulty in Breathing	Decreased Fetal Movements
34	SK	1424983	CASES	30	Primigravida	3rd	23-08-2022	30-05-2023	33	No	No	No	No	No	No	No	Yes
35	R	1499808	CASES	24	Primigravida	3rd	04-08-2022	11-05-2023	36	Yes	No	No	No	Yes	No	No	No
36	C	1512664	CASES	28	Multigravida	2nd	15-12-2022	10-05-2023	22	No	No	No	No	Yes	No	No	No
37	SK	1521152	CASES	24	Primigravida	3rd	29-08-2022	05-06-2023	37	No	No	No	No	Yes	No	No	No
38	T	1198102	CASES	25	Primigravida	3rd	31-03-2022	01-07-2023	35	No	No	Yes	Yes	No	No	No	No
39	JL	1413575	CASES	26	Multigravida	3rd	10-04-2022	17-01-2023	32	No	Yes	No	No	No	No	No	No
40	SP	1401462	CASES	28	Multigravida	3rd	03-06-2022	12-11-2022	32	No	No	No	No	No	No	No	No
41	RKs	1446014	CASES	38	Multigravida	2nd	30-08-2022	06-06-2023	19	No	No	No	No	Yes	No	No	No
42	Sk	740982	CASES	27	Multigravida	3rd	13-02-2022	20-11-2022	37	No	No	No	No	No	No	No	Yes
43	RK	1407631	CASES	24	Primigravida	3rd	02-10-2022	17-11-2022	37	No	No	No	No	No	No	No	Yes
44	SD	1446759	CASES	44	Primigravida	3rd	05-10-2022	14-02-2023	35	No	No	No	No	No	No	No	Yes
45	JR	1476854	CASES	38	Multigravida	2nd	15-09-2022	22-06-2023	24	No	No	No	No	No	Yes	No	No
46	Pp	1444235	CASES	27	Primigravida	1st	13-11-2022	20-08-2023	8	No	No	Yes	No	Yes	No	No	No
47	A	1230005	CASES	24	Multigravida	3rd	07-02-2022	04-08-2023	37	No	No	No	No	Yes	No	No	No
48	KP	1361121	CASES	39	Multigravida	3rd	21-04-2022	28-01-2023	36	No	No	No	No	No	No	No	Yes
49	AS	39163	CASES	37	Multigravida	3rd	23-07-2022	30-04-2023	30	No	No	No	No	Yes	No	No	No
50	RD	1468383	CASES	45	Multigravida	2nd	08-11-2022	18-05-2023	27	No	No	No	No	Yes	No	No	No
51	R	1512619	CASES	23	Primigravida	3rd	08-08-2022	15-05-2023	38	No	No	No	No	No	No	No	Yes
52	SK	1512684	CASES	30	Primigravida	3rd	01-08-2022	08-05-2023	38	No	No	No	Yes	No	No	No	No
53	RM	1277379	CASES	38	Primigravida	3rd	24-10-2022	31-07-2023	28	Yes	No	No	No	Yes	No	No	No
54	NS	971892	CASES	28	Primigravida	2nd	18-07-2022	25-04-2023	15	No	No	Yes	No	No	No	No	No
55	SG	1532377	CASES	28	Primigravida	3rd	24-09-2022	01-07-2023	36	Yes	No	No	No	Yes	No	No	No
56	SK	242117	CASES	36	Multigravida	3rd	24-09-2022	11-07-2023	35	Yes	No	No	No	Yes	No	No	No
57	A	1419355	CASES	24	Primigravida	3rd	09-09-2023	15-06-2023	38	No	No	No	No	Yes	No	No	No
58	NT	1135509	CASES	28	Primigravida	3rd	14-02-2022	21-11-2022	39	No	No	No	No	Yes	No	No	No
59	HK	1361459	CASES	24	Multigravida	3rd	28-04-2022	02-02-2023	37	No	No	No	No	No	Yes	No	No
60	KK	1345333	CASES	33	Multigravida	3rd	25-04-2022	01-02-2023	35	No	No	No	No	Yes	No	No	No
61	PS	1443933	CASES	30	Multigravida	3rd	18-05-2022	25-02-2023	33	Yes	No	No	No	No	No	No	No
62	PS	742569	CASES	29	Multigravida	3rd	13-04-2022	20-01-2023	37	Yes	No	No	No	Yes	No	No	No
63	LS	1427605	CASES	28	Multigravida	3rd	01-09-2022	18-06-2023	38	No	No	No	No	No	No	No	Yes
64	SP	1287930	CASES	33	Multigravida	3rd	15-09-2022	22-06-2023	35	No	No	No	No	No	No	No	Yes
65	T	1427590	CASES	28	Multigravida	3rd	03-09-2022	10-06-2023	36	No	No	No	No	Yes	No	No	No
66	SK	1438797	CASES	26	Multigravida	3rd	16-06-2022	23-03-2023	26	No	Yes	No	No	No	No	No	No
67	JK	1531658	CASES	37	Multigravida	1st	13-03-2023	20-12-2023	10	Yes	Yes	Yes	Yes	No	No	No	No
68	M	1394918	CASES	28	Primigravida	3rd	05-02-2022	12-11-2022	37	No	No	No	No	Yes	No	No	No
69	RK	22073	CASES	35	Multigravida	3rd	25-09-2022	02-07-2023	34	No	No	No	No	No	No	No	Yes

SR.No.	Bleeding Per Vaginum/ Spotting	Pallor	Icterus	Hepatomegaly	Ascitis	Increased BP readings	ICP	Hyperemesis Gravidarum	Acute Liver Failure	HELLP	Acute Fatty Liver of Pregnancy	Pre eclampsia	Acute on Chronic Liver Failure	Drug Induced Liver Injury	Hb	TLC	Platelet count	AST	ALT
34	No	Yes	No	No	No	No	Moderate								8.3	6.8	206	161	152
35	No	No	No	No	No	No	Mild								9.4	13.82	235	57	114
36	No	No	No	No	No	No									11.1	18.19	258	206	192
37	Yes	Yes	No	No	No	No									11.8	15.1	100	100	129
38	No	No	Yes	No	No	No	Moderate								8.8	11.6	200	73	46
39	No	No	No	No	No	No									11.2	11.5	50	98	25
40	Yes	Yes	No	No	No	No									9.5	6.61	110	342	321
41	No	No	No	No	No	No	Moderate								7.9	19.6	275	72	121
42	No	No	Yes	No	No	No									9.3	5.9	186	233	250
43	No	No	No	No	No	No	Mild								10.6	7.5	160	103	116
44	No	No	No	No	No	Yes				Yes					12.5	8.3	150	75	127
45	Yes	No	No	No	No	No	Mild								8.1	15.26	194	130	104
46	No	No	No	No	No	No		Yes							10.5	2.4	104	137	60
47	No	No	No	No	No	No	Mild								10.6	11.1	134	460	531
48	No	No	No	No	No	No						Yes			8.9	12.3	320	73	92
49	Yes	No	No	No	No	No	Moderate								9.3	8.82	160	235	290
50	Yes	No	No	No	No	No	Moderate								10.2	7.9	204	109	236
51	No	No	Yes	No	No	No	Severe					Yes			12.6	20.1	197	295	420
52	Yes	No	Yes	No	No	No									9	36	25	405	250
53	Yes	No	No	No	No	No	Severe								11.1	13.3	158	364	480
54	No	No	No	No	No	No		Yes							12.2	8.27	277	154	193
55	No	No	No	No	No	No	Moderate								12.1	11.2	248	414	749
56	No	No	No	No	No	No	Mild								9.7	13.4	246	103	115
57	No	No	No	No	No	No	Mild								13.8	11.1	142	183	198
58	No	No	No	No	No	No									10.3	5.9	154	45	102
59	No	No	No	No	No	No									11	9.35	181	65	100
60	No	No	No	No	No	No									10.5	10.6	157	123	140
61	No	No	No	No	No	Yes									11.9	10.3	146	89	69
62	No	No	No	No	No	No									12.3	9.5	306	85	99
63	No	No	No	No	No	No									12.1	8.83	280	126	195
64	No	No	No	No	No	No									10.6	15.3	148	180	218
65	No	No	No	No	No	No	Mild								11.6	8.69	152	329	629
66	No	No	No	No	No	No									10.1	14.1	280	221	135
67	No	No	Yes	Yes	Yes	No							Yes		9.7	13.7	150	754	326
68	No	No	No	No	No	No									10.4	9.92	152	71	97
69	No	No	No	No	No	No									9.2	13.5	260	140	160

SR.No.	ALP	Bile acids	Total Bilirubin	D. Bilirubin	Total Protein	S. Albumin	HIV	HAV	HEV	HBsAg	HCV	Dengue	Leptospirosis	Scrub typhus	Mode of delivery	Indications Of Cesarean Section	Fetal GENDER	Fetal Weight
34	285	81.4	0.42	0.22	6.8	3.69	NR	NA	NA	NR	NR	Negative	Negative	Negative	LSCS	Fetal distress	Male	2.02
35	77	33.9	0.46	0.23	7.2	4.63	NR	NA	NA	NR	NR	Negative	Negative	Negative	LSCS	Fetal distress	Female	2.8
36	121		1.25	0.75	6.4	3.5	NR	NA	NA	NR	NR	Negative	Negative	Negative	Preterm NVD		Abortion	0.62
37	691	6	0.98	0.17	6.4	3.16	NR	NA	NA	NR	NR	Negative	Negative	Negative	Term NVD		Male	2.64
38	447	48.6	5.11	2.68	7.1	3.48	NR	NR	NR	NR	NR	Negative	Negative	Negative	LSCS	Moderate ICP	Male	2.5
39	335		1.23	0.73	6.4	2.84	NR	NA	NA	NR	NR	Positive	Negative	Negative	LSCS	Fetal distress	Female	2.5
40	307		0.35	0.1	6.1	3.03	NR	NR	NR	NR	NR	Negative	Negative	Negative	LSCS	Fetal distress	Male Female	1.9,1.0
41	187	41.2	0.17	0.08	7	3.63	NR	NR	NR	NR	NR	Negative	Negative	Negative	Term NVD		Male	0.32
42	299		4.2	3.11	7.3	3.91	NR	NR	NR	NR	NR	Negative	Negative	Negative	LSCS	Fetal distress	Male	3.1
43	222	26.8	0.46	0.23	6.6	3.82	NR	NR	NR	NR	NR	Negative	Negative	Negative	Term NVD	Mild ICP	Male	2.52
44	251	17.5	0.42	0.13	5.2	2.5	NR	NR	NR	NR	NR	Negative	Negative	negative	LSCS	Fetal distress	Male	1.98
45	378	22.4	1.32	0.74	5.9	2.79	NR	NR	NR	NR	NR	Negative	Negative	Negative	LSCS	Fetal distress	Male Male	0.645
46	104		0.32	0.08	6.2	3.26	NR	NA	NA	NR	NR	positive	Negative	Negative	LSCS	Fetal distress	Male	2.7
47	199	23.8	0.94	0.66	6.4	3.42	NR	NR	NR	NR	NR	Negative	Negative	Negative	LSCS	Mild ICP	Female	2.5
48	236		0.35	0.07	6.6	3.29	NR	NA	NA	NR	NR	Negative	Negative	Negative	LSCS	Fetal distress	Female	2.8
49	198	66.8	0.8	0.51	6.6	3.57	NR	NA	NA	NR	NR	Negative	Negative	Negative	LSCS	Moderate ICP	Male	3
50	269	70.5	0.37	0.27	6.1	3.05	NR	NA	NA	NR	NR	Negative	Negative	Negative	LSCS	Fetal distress	Female Female	1.9,2.2
51	373	152	3.76	2.92	5.8	2.89	NR	NA	NA	NR	NR	Negative	Negative	Negative	LSCS	Pre eclampsia	Male	2.3
52	158		5.95	5.04	4.8	2.25	NR	NA	NA	NR	NR	Negative	Negative	Negative	LSCS	Previous LSCS	Male	2.5
53	224		0.46	0.11	6.5	3.56	NR	NA	NA	NR	NR	Negative	Negative	Negative	LSCS	Fetal distress	Female	1.48
54	63		0.99	0.39	6.8	3.9	NR	NA	NA	NR	NR	Negative	Negative	Negative	Term NVD		Male	2.4
55	233	56.5	0.56	0.34	5.7	2.86	NR	NA	NA	NR	NR	Negative	Negative	Negative	LSCS	Fetal distress	Female	2.5
56	259	21.8	0.38	0.08	7	3.12	NR	NA	NA	NR	NR	Negative	Negative	Negative	LSCS	Fetal distress	Female	3.3
57	536	19.2	0.58	0.15	7.1	3.55	NR	NA	NA	NR	NR	Negative	Negative	Negative	LSCS	Fetal distress	Male	2.5
58	271	14.2	0.22	0.12	6.5	3.26	NR	NA	NA	NR	NR	Negative	Negative	Negative	Term NVD		Male	2.62
59	196		0.28	0.05	6.4	3.29	NR	NA	NA	NR	NR	Negative	Negative	Negative	Term NVD		Female	3.02
60	156		0.8	0.2	6.5	3.8	NR	NA	NA	NR	NR	Negative	Negative	Negative	LSCS	Fetal distress	Male	2.1
61	160		0.49	0.09	4.6	1.98	NR	NA	NA	NR	NR	Negative	Negative	Negative	LSCS	Unfavourable cervix	Male Female	1.8,2
62	337		0.74	0.55	6.7	3.45	NR	NA	NA	NR	NR	Negative	Negative	Negative	LSCS	Fetal distress	Female	3.36
63	290	11.5	0.35	0.2	6.6	3.64	NR	NA	NA	NR	NR	Negative	Negative	Negative	LSCS	Scar tenderness	Male	2.9
64	182	12.1	0.11	0.04	6.5	3.71	NR	NA	NA	NR	NR	Negative	Negative	Negative	LSCS	Scar tenderness	Male	2.8
65	227	22.2	1	0.22	6.6	3.22	NR	NA	NA	NR	NR	Negative	Negative	Negative	LSCS	Failure of induction	Male	3.44
66	471		1.46	0.64	6.6	2.95	NR	NA	NA	NR	NR	Negative	Negative	Negative	LSCS	Fetal distress	Male	1.16
67	149		32.48	24.99	9.3	2.91	NR	NA	NA	NR	REACTIVE	Negative	Positive	Negative	LSCS	ACLF	Female	1.3
68	185		0.26	0.18	6.8	3.54	NR	NA	NA	NR	NR	Negative	Negative	Negative	LSCS	Fetal distress	Male	2.86
69	167		1.09	1	6.8	3.5	NR	NA	NA	NR	NR	Negative	Negative	Negative	LSCS	Fetal distress	Female	2.46

SR.No.	Fetal Outcome	Fetal complications (If Any)	Maternal Outcome	Maternal Complications (If Any)	Disease
34	Fetal Growth restriction		Normal	Anemia	Moderate ICP
35	Normal Live Birth		Normal		Mild ICP
36	Abortion		Normal		
37	Normal Live Birth		Normal		
38	Normal Live Birth		Prolonged Hospital stay		Moderate ICP
39	Preterm birth	Prematurity	Normal		Tropical fever
40	Intrauterine Death,pre term birth	Prematurity	Normal		
41	Abortion		Normal		Moderate ICP
42	Normal Live Birth		Normal		
43	Intrauterine Death	NICU admission	Normal		Mild ICP
44	Normal Live Birth		Prolonged Hospital stay	HELLP	HELLP
45	Intrauterine Death		Prolonged Hospital stay		Mild ICP
46	Normal Live Birth		Prolonged Hospital stay	Sepsis	Hyperemesis Gravidarum,Tropical fever
47	Normal Live Birth		Normal		Mild ICP
48	Normal Live Birth		Normal	Pre -eclampsia	Pre eclampsia
49	Normal Live Birth		Prolonged Hospital stay		Moderate ICP
50	Preterm birth	Prematurity	Prolonged Hospital stay		Moderate ICP
51	Normal Live Birth		Normal	Pre -eclampsia	Pre eclampsia
52	Normal Live Birth		Prolonged Hospital stay	Acute kidney injury,Sepsis,Septic shock	
53	Preterm birth	Prematurity	Normal		Severe ICP
54	Preterm birth	Prematurity	Normal		Hyperemesis Gravidarum
55	Normal Live Birth		Prolonged Hospital stay		Moderate ICP
56	Normal Live Birth		Prolonged Hospital stay		Mild ICP
57	Normal Live Birth		Normal		Mild ICP
58	Normal Live Birth		Normal		
59	Normal Live Birth		Normal		
60	Normal Live Birth		Normal		
61	Preterm birth	Prematurity	Prolonged Hospital stay		
62	Normal Live Birth		Normal		
63	Normal Live Birth		Normal		
64	Normal Live Birth		Normal		
65	Normal Live Birth		Normal		Mild ICP
66	Fetal Growth restriction		Normal		
67	Preterm birth	Prematurity	Mortality	Acute Liver Failure	Acute on Chronic Liver Disease
68	Preterm birth	Prematurity	Normal		
69	Preterm birth	Prematurity	Normal		

SR.No.	Name	MRD No.	GROUP	Age (years)	Gravida	Trimester	LMP	EDD	Gestation Age (weeks)	Co Morbidities	Fever	Persistent Nausea/ Vomiting	Yellowish Discoloration	Pain Abdomen	Itching	Difficulty in Breathing	Decreased Fetal Movements
70	J	407897	CASES	33	Primigravida	3rd	04-02-2022	11-11-2022	37	No	No	No	No	Yes	No	No	No
71	TS	1291930	CASES	25	Primigravida	3rd	29-01-2022	06-11-2022	36	No	No	No	No	Yes	No	No	No
72	M	723410	CASES	29	Multigravida	3rd	14-02-2022	24-10-2022	37	No	No	No	No	No	No	No	No
73	S	1398347	CASES	32	Multigravida	3rd	02-02-2022	09-11-2022	36	No	No	No	No	No	No	No	No
74	SR	1361623	CASES	36	Primigravida	3rd	09-02-2022	16-11-2022	36	No	No	No	No	No	No	No	Yes
75	C	1398326	CASES	22	Multigravida	2nd	24-04-2022	29-01-2023	25	No	No	No	No	No	No	Yes	No
76	PV	1399057	CASES	33	Multigravida	3rd	10-02-2022	17-11-2022	35	No	No	No	No	Yes	No	No	No
77	R	1404477	CASES	37	Multigravida	3rd	09-03-2022	16-12-2022	34	No	No	No	No	No	No	No	No
78	JK	202100	CASES	35	Multigravida	3rd	22-07-2022	29-04-2023	37	Yes	No	No	No	No	No	No	No
79	HK	1479171	CASES	36	Multigravida	2nd	26-09-2022	03-07-2023	23	Yes	No	No	No	No	No	No	No
80	S	1131959	CASES	38	Multigravida	3rd	13-05-2022	21-01-2023	31	No	No	No	No	No	No	No	Yes
81	Jk	1566938	CASES	32	Primigravida	3rd	15-02-2022	24-11-2023	30	No	No	No	Yes	No	Yes	No	No
82	RK	1545644	CASES	28	Primigravida	3rd	31-10-2022	07-08-2023	36	No	No	No	No	No	No	No	Yes
83	SK	1554305	CASES	40	Multigravida	3rd	16-10 2022	23-07-2023	39	Yes	No	No	No	No	No	No	No
84	EJ	1550990	CASES	34	Multigravida	3rd	12-11-2022	19-08-2023	35	Yes	No	No	No	Yes	Yes	No	No
85	MK	1517148	CASES	30	Multigravida	3rd	26-11-2022	03-08-2023	36	Yes	No	No	No	Yes	No	No	No
86	K	1594155	CASES	31	Primigravida	3rd	19-12-2022	26-09-2023	36	No	No	Yes	Yes	Yes	Yes	No	Yes
87	HK	1573834	CASES	30	Multigravida	3rd	08-12-2022	15-09-2023	36	No	No	No	No	No	No	No	Yes
88	SD	1437621	CASES	24	Primigravida	2nd	02-10-2022	10-07-2023	24	No	No	No	No	Yes	Yes	No	No
89	S	1550003	CASES	40	Multigravida	3rd	05-11-2022	12-08-2023	36	Yes	No	No	No	Yes	No	No	No
90	SM	1607834	CASES	33	Multigravida	3rd	07-03-2023	14-12-2023	31	No	No	No	No	Yes	No	No	No
91	Kk	1617162	CASES	38	Multigravida	2nd	26-06-2023	03-04-2024	18	No	No	Yes	No	No	No	No	No
92	S	1639548	CASES	27	Multigravida	2nd	20-07-2023	27-04-2024	21	Yes	No	No	Yes	Yes	Yes	No	No
93	SS	1526083	CASES	37	Multigravida	2nd	10-12-2022	16-09-2023	28	No	No	No	Yes	No	No	No	No
94	P	1450532	CASES	23	Multigravida	3rd	15-06-2022	31-01-2023	32	No	No	No	Yes	Yes	Yes	No	No
95	PK	1405469	CASES	29	Multigravida	3rd	26-03-2022	31-12-2022	36	No	No	No	No	No	No	No	No
96	RG	945707	CASES	32	Multigravida	1st	28-08-2022	04-06-2023	8	No	Yes	No	No	No	No	No	No
97	K	1485539	CASES	29	Primigravida	3rd	25-12-2022	01-10-2023	38	Yes	Yes	No	No	No	Yes	No	No
98	DK	1432377	CASES	35	Multigravida	3rd	20-11-2022	27-08-2023	34	No	No	No	No	Yes	No	No	No
99	Dj	1556777	CASES	27	Multigravida	3rd	23-10-2022	30-07-2023	32	No	No	No	No	Yes	Yes	No	No
100	RK	1457245	CASES	25	Multigravida	3rd	22-06-2022	29-03-2023	32	No	No	No	No	Yes	No	No	Yes

SR.No.	Bleeding Per Vaginum/ Spotting	Pallor	Icterus	Hepatomegaly	Ascitis	Increased BP readings	ICP	Hyperemesis Gravidarum	Acute Liver Failure	HELLP	Acute Fatty Liver of Pregnancy	Pre eclampsia	Acute on Chronic Liver Failure	Drug Induced Liver Injury	Hb	TLC	Platelet count	AST	ALT
70	No	No	No	No	No	No									9.5	13.5	289	98	96
71	No	No	No	No	No	No	Mild								11.1	9.2	160	112	104
72	No	No	No	No	No	No									11.5	7.1	140	48	102
73	Yes	Yes	No	No	No	No									10.2	9.1	221	109	117
74	No	No	No	No	No	No								Yes	11.3	8.3	136	133	119
75	No	No	No	No	No	No				Yes					9.7	17.1	118	97	176
76	No	No	No	No	No	No									10.9	12.7	150	157	279
77	Yes	No	No	No	No	No	Mild								10.8	8.3	177	92	82
78	No	No	No	No	No	No	Mild								13.3	7.1	164	116	184
79	Yes	No	No	No	No	No	Mild								9.5	10.2	295	108	144
80	No	No	No	No	No	No									11.3	8.9	237	59	98
81	Yes	No	Yes	No	Yes	No	Severe								9.3	12.3	152	118	47
82	Yes	Yes	No	No	No	No						Yes			10.5	14.8	165	334	258
83	No	No	No	No	No	Yes	Mild								12.2	16	246	221	315
84	No	No	No	No	No	No	Moderate								9.7	17.91	253	159	144
85	No	No	No	No	No	No									11.8	9.6	224	51	114
86	Yes	Yes	Yes	Yes	Yes	No	Severe				Yes				9.8	14.7	38	2450	536
87	No	No	No	No	No	No	Moderate								10.6	10.7	32	144	60
88	No	No	No	No	No	No	Moderate								13	7.11	138	277	289
89	No	No	No	No	No	No									9.6	10.7	212	220	275
90	No	Yes	Yes	Yes	No	No					Yes				10.3	12.5	277	160	190
91	No	No	No	No	Yes	No	Mild								8.2	10.2	48	287	122
92	No	No	Yes	Yes	No	No	Severe						Yes		7.6	9	188	710	437
93	No	No	Yes	Yes	No	No			Yes					Yes	8.6	21.3	338	210	390
94	No	Yes	Yes	No	No	No	Severe		Yes						9.8	20.6	168	1956	1298
95	Yes	Yes	No	No	No	No	Mild								11.2	16.7	111	143	124
96	Yes	No	No	No	No	No									13.1	5.2	155	96	90
97	Yes	No	No	No	No	No	Severe								9.2	10.7	178	131	195
98	No	No	No	No	No	No									12.2	9.9	105	129	145
99	No	No	No	No	No	No	Moderate								8.4	4.6	168	286	363
100	Yes	No	No	No	No	Yes						Yes			13	11.3	150	232	345

SR.No.	ALP	Bile acids	Total Bilirubin	D. Bilirubin	Total Protein	S. Albumin	HIV	HAV	HEV	HBsAg	HCV	Dengue	Leptospirosis	Scrub typhus	Mode of delivery	Indications Of Cesarean Section	Fetal GENDER	Fetal Weight
70	327		0.32	0.05	6.5	3.34	NR	NA	NA	NR	NR	Negative	Negative	Negative	LSCS	Fetal distress	Male	2.76
71	221		0.79	0.34	7.1	4.14	NR	NA	NA	NR	NR	Negative	Negative	Negative	LSCS	Fetal distress	Male	2.86
72	126		0.28	0.18	5.2	2.8	NR	NA	NA	NR	NR	Negative	Negative	Negative	LSCS	Fetal distress	Male	2.6
73	240		0.69	0.16	7.1	3.27	NR	NA	NA	NR	NR	Negative	Negative	Negative	LSCS	Type IV placenta previa	Male	2.1
74	131		0.29	0.09	6	3.57	NR	NA	NA	NR	NR	Negative	Negative	Negative	LSCS	Fetal distress	Male	2.1
75	157		0.25	0.16	6	3.07	NR	NA	NA	NR	NR	Negative	Negative	Negative	LSCS	Deteriorating Maternal condition	Male	0.86
76	258		0.26	0.08	7.1	3.78	NR	NA	NA	NR	NR	Negative	Negative	Negative	LSCS	Scar tenderness	Female	2.28
77	215	20.7	0.42	0.28	6.1	3.41	NR	NA	NA	NR	NR	Negative	Negative	Negative	Classical C-Section	Previous LSCS	Male	2.58
78	136	19.6	0.4	0.07	6.4	3.36	NR	NA	NA	NR	NR	Negative	Negative	Negative	LSCS	Scar tenderness	Male	3.6
79	317	30.3	0.23	0.2	5.9	2.86	NR	NA	NA	NR	NR	Negative	Negative	Negative	Classical C-Section	Previous LSCS	Male	1.2
80	153		0.35	0.07	6.4	3.06	NR	NA	NA	NR	NR	Negative	Negative	Negative	LSCS	Type IV placenta previa	Female	1.9
81	271	289.7	21.18	16.38	5.7	2.94	NR	NR	NR	NR	NR	Negative	Negative	Negative	LSCS	Severe ICP	Female	1.38
82	275		0.3	0.16	6.5	2.75	NR	NA	NA	NR	NR	Negative	Negative	Negative	Preterm NVD		Male	1.9
83	226	22.6	0.47	0.09	6.2	2.99	NR	NA	NA	NR	NR	Negative	Negative	Negative	LSCS	Previous LSCS	Female	3.5
84	300	52.9	0.6	0.3	5.9	3.6	NR	NA	NA	NR	NR	Negative	Negative	Negative	LSCS	Moderate ICP	Female	2.2
85	142	7.9	0.3	0.2	6.6	3.55	NR	NA	NA	NR	NR	Negative	Negative	Negative	LSCS	Fetal distress	Male	1.9
86	251	118.1	5.86	4.89	5.9	2.82	NR	NR	NR	NR	NR	positive	Negative	Negative	LSCS	Severe ICP	Male	1.92
87	245	40.8	0.61	0.33	6.1	3.52	NR	NA	NA	NR	NR	positive	Negative	Negative	LSCS	Moderate ICP	Female	2.5
88	303	64.3	0.83	0.2	6.6	3.24	NR	NA	NA	NR	NR	Negative	Negative	Negative	Term NVD	Moderate ICP	Female	3.2
89	279	16.9	0.53	0.11	6.9	3.3	NR	NA	NA	NR	NR	Negative	Negative	Negative	LSCS	Acute fatty liver of pregnancy	Male	2.24
90	283		0.83	0.5	6	3.9	NR	NA	NA	NR	NR	Negative	Negative	Negative	LSCS	Scar tenderness	Female	1.8
91	342	29	0.76	0.38	5.9	2.78	NR	NA	NA	NR	NR	Positive	Negative	Negative	LSCS	Mild ICP	Male	2.5
92	159	268	10	8.6	5.9	2.9	NR	NA	NA	NR	NR	Negative	Negative	Negative	LSCS	ACLF	Male	3.2
93	127		23	20	4.7	2.08	NR	NR	NR	NR	NR	Negative	Negative	Negative	Preterm NVD		Female	0.92
94	387	293	9.31	7.7	4.3	2.12	NR	NA	REACTIVE	NR	NR	Negative	Negative	Negative	LSCS	Severe ICP	Female	1.6
95	160	29	0.9	0.5	6	3.9	NR	NA	NA	NR	NR	Positive	Negative	Negative	LSCS	Type IV Placenta previa	Female	2.5
96	140		0.48	0.39	6.1	3.28	NR	NA	NA	NR	NR	Positive	Positive	Negative	LSCS	Failure of induction	Male Male	1.8,1.7
97	232	226	0.41	0.11	6.7	3.46	NR	NA	NA	NR	NR	Negative	Negative	Negative	Term NVD		Female	3.2
98	238		1.49	0.95	5.2	2.91	NR	NA	NA	NR	NR	Negative	Negative	Negative	LSCS	Fetal distress	Male	3.4
99	139	68.6	0.76	0.55	5.8	2.91	NR	NA	NA	NR	NR	Negative	Negative	Negative	LSCS	Moderate ICP	Male	2.7
100	112	3.7	0.32	0.07	6.1	3.27	NR	NR	NR	NR	NR	Negative	Negative	Negative	LSCS	Pre eclampsia	Male	1.5

SR.No.	Fetal Outcome	Fetal complications (If Any)	Maternal Outcome	Maternal Complications (If Any)	Disease
70	Preterm birth	Prematurity	Normal		
71	Preterm birth	Prematurity	Normal		Mild ICP
72	Preterm birth	Prematurity	Normal		
73	Fetal Growth restriction	NICU admission	Prolonged Hospital stay		
74	Fetal Growth restriction	NICU admission	Normal		Drug Induced Liver Injury
75	Intrauterine Death		Prolonged Hospital stay	APH,HELLP	HELLP
76	Preterm birth	Perinatal Asphyxia	Normal		
77	Preterm birth	NICU admission	Prolonged Hospital stay	Obstetrical hysterectomy	Mild ICP
78	Preterm birth	Prematurity	Normal		Mild ICP
79	Fetal Growth restriction	Prematurity	Prolonged Hospital stay	APH,Wound sepsis	Mild ICP
80	Intrauterine Death		Normal		
81	Preterm birth	Prematurity	Prolonged Hospital stay		Severe ICP
82	Intrauterine Death		Multiorgan failure	Pre -eclampsia	Pre-eclampsia
83	Normal Live Birth		Normal		Mild ICP
84	Normal Live Birth		Normal		Moderate ICP
85	Preterm birth	Prematurity	Prolonged Hospital stay		
86	Preterm birth	Prematurity	Prolonged Hospital stay		Acute fatty liver of pregnancy,Tropical fever
87	Preterm birth	Prematurity	Prolonged Hospital stay		Tropical fever
88	Preterm birth	Prematurity	Normal		Moderate ICP
89	Preterm birth	Prematurity	Prolonged Hospital stay		
90	Preterm birth	Prematurity	Prolonged Hospital stay		
91	Preterm birth	Prematurity	Normal		Tropical fever
92	Preterm birth	Prematurity	Prolonged Hospital stay	Anemia	Acute on Chronic Liver Failure
93	Intrauterine Death		Prolonged Hospital stay	Acute Liver Failure	Acute Liver Failure,Drug induced Liver Injury
94	Fetal Growth restriction		Prolonged Hospital stay	Acute Liver Failure	Acute Viral Hepatitis,Acute Liver Failure,severe ICP
95	Normal Live Birth		Prolonged Hospital stay	APH,Obstetrical Hysterectomy	Mild ICP
96	Fetal Growth restriction		Prolonged Hospital stay		Tropical fever
97	Normal Live Birth		Normal		Severe ICP
98	Normal Live Birth		Normal		
99	Preterm birth	Prematurity	Normal		Moderate ICP
100	Preterm birth	Prematurity	Normal	Pre -eclampsia	Pre- eclampsia

SR.No.	Name	MRD No.	GROUP	Age (years)	Gravida	Trimester	LMP	EDD	Gestation Age (weeks)	Co Morbidities	Fever	Persistent Nausea/ Vomiting	Yellowish Discoloration	Pain Abdomen	Itching	Difficulty in Breathing	Decreased Fetal Movements
1	RS	1441467	CONTROL	35	Multigravida	3rd	18-05-2022	25-02-2023	33	No	No	No	No	No	No	No	No
2	BS	1426067	CONTROL	23	Primigravida	3rd	05-05-2022	09-02-2023	30	No	No	No	No	No	No	No	No
3	S	1299722	CONTROL	25	Primigravida	3rd	04-04-2022	11-01-2023	35	No	No	No	No	Yes	No	No	No
4	MK	1401194	CONTROL	31	Multigravida	3rd	20-04-2022	27-01-2023	35	No	No	No	No	Yes	Yes	No	No
5	N	1503953	CONTROL	24	Multigravida	3rd	08-10-2022	15-07-2023	28	No	No	No	No	Yes	No	No	No
6	GK	298497	CONTROL	33	Multigravida	3rd	28-04-2022	02-02-2023	38	No	No	No	No	Yes	No	No	No
7	MS	1346609	CONTROL	27	Primigravida	3rd	18-08-2022	25-05-2023	38	No	No	No	No	No	No	No	Yes
8	A	1292077	CONTROL	25	Primigravida	3rd	05-02-2022	12-11-2022	37	No	No	No	No	No	No	No	No
9	SR	1480077	CONTROL	32	Multigravida	3rd	01-09-2022	08-06-2023	35	No	No	No	No	No	No	No	Yes
10	IR	1515301	CONTROL	26	Multigravida	3rd	30-08-2022	06-06-2023	36	No	No	No	No	Yes	No	No	No
11	JK	62521	CONTROL	29	Multigravida	3rd	29-05-2022	05-03-2023	37	No	No	No	No	Yes	No	No	No
12	P	1356470	CONTROL	28	Primigravida	3rd	05-05-2022	12-02-2023	37	No	No	No	No	Yes	No	No	No
13	P	911148	CONTROL	41	Multigravida	3rd	25-04-2022	02-02-2023	39	No	No	No	No	Yes	No	No	No
14	PJ	1527375	CONTROL	28	Primigravida	3rd	10-09-2022	17-06-2023	37	No	No	No	No	No	No	No	Yes
15	GK	1495028	CONTROL	39	Multigravida	3rd	11-09-2022	18-06-2023	38	No	No	No	No	Yes	No	No	No
16	U	391300	CONTROL	32	Multigravida	3rd	03-12-2022	09-09-2023	38	No	No	No	No	No	No	No	No
17	K	1442102	CONTROL	29	Multigravida	3rd	20-04-2022	25-01-2023	37	No	No	No	No	Yes	No	No	No
18	S	422377	CONTROL	32	Multigravida	3rd	25-07-2022	02-05-2023	40	No	No	No	No	Yes	No	No	No
19	SS	872254	CONTROL	29	Multigravida	3rd	22-08-2022	29-05-2023	37	No	No	No	No	Yes	No	No	No
20	A	1463933	CONTROL	29	Multigravida	3rd	01-06-2022	08-03-2023	38	No	No	No	No	Yes	No	No	No
21	EG	279736	CONTROL	34	Multigravida	3rd	06-07-2022	13-04-2023	37	No	No	No	No	Yes	No	No	No
22	AJ	1435985	CONTROL	30	Primigravida	3rd	12-06-2022	19-03-2023	38	No	No	No	No	Yes	No	No	No
23	K	1088679	CONTROL	24	Multigravida	3rd	18-07-2022	24-04-2023	40	No	No	No	No	Yes	No	No	No
24	CJ	987401	CONTROL	31	Multigravida	3rd	11-07-2022	18-04-2023	37	No	No	No	No	Yes	No	No	No
25	KK	419842	CONTROL	32	Multigravida	3rd	24-05-2022	28-02-2023	32	No	No	No	No	Yes	No	No	No
26	A	1292077	CONTROL	25	Primigravida	3rd	05-02-2022	12-11-2022	37	No	No	No	No	Yes	No	No	No
27	K	737985	CONTROL	27	Primigravida	3rd	25-05-2022	02-03-2023	39	No	No	No	No	Yes	No	No	No
28	HK	1437300	CONTROL	30	Multigravida	3rd	07-11-2022	14-08-2023	37	No	No	No	No	Yes	No	No	No
29	PD	1016841	CONTROL	34	Multigravida	2nd	10-04-2023	17-01-2024	24	No	No	No	No	No	No	No	No
30	D	1280617	CONTROL	26	Primigravida	3rd	24-04-2022	3101/2023	36	No	No	No	No	Yes	No	No	No
31	SM	746079	CONTROL	29	Multigravida	3rd	05-04-2022	12-01-2023	38	No	No	No	No	No	No	No	Yes
32	IK	1641433	CONTROL	36	Primigravida	3rd	02-04-2023	09-01-2024	37	Yes	No	No	No	Yes	No	No	No
33	UR	593962	CONTROL	31	Multigravida	3rd	11-04-2023	18-01-2024	36	No	No	No	No	No	No	No	Yes
34	N	1641913	CONTROL	24	Multigravida	3rd	23-03-2023	30-12-2023	39	No	No	No	No	No	No	No	No
35	RM	1641794	CONTROL	29	Multigravida	3rd	22-03-2023	29-12-2023	39	No	No	No	No	Yes	No	No	No
36	AT	1555804	CONTROL	29	Primigravida	3rd	14-03-2023	21-12-2023	37	No	No	No	No	Yes	No	No	No
37	S	1592874	CONTROL	34	Multigravida	3rd	27-02-2023	05-12-2023	40	No	No	No	No	Yes	No	No	No
38	KK	1578068	CONTROL	29	Multigravida	3rd	02-12-2022	09-09-2023	38	Yes	No	No	No	No	No	No	Yes
39	LK	308600	CONTROL	31	Primigravida	3rd	17-02-2023	24-11-2023	39	Yes	No	No	No	Yes	No	No	No
40	AS	184512	CONTROL	32	Multigravida	3rd	05-01-2023	12-10-2023	37	No	No	No	No	Yes	No	No	No
41	KS	1516208	CONTROL	30	Multigravida	3rd	06-04-2023	13-01-2024	36	Yes	No	No	No	Yes	No	No	No
42	MK	9098	CONTROL	34	Multigravida	3rd	28-03-2023	05-01-2024	39	No	No	No	No	Yes	No	No	No
43	A	1188857	CONTROL	26	Multigravida	3rd	22-11-2022	29-08-2023	38	No	No	No	No	Yes	No	No	No
44	R	1518798	CONTROL	34	Multigravida	3rd	17-03-2023	24-12-2023	37	Yes	No	No	No	No	No	No	No
45	Ps	1454827	CONTROL	36	Multigravida	3rd	24-12-2022	01-10-2023	38	Yes	No	No	No	Yes	No	No	No
46	U	1589236	CONTROL	27	Primigravida	3rd	25-12-2022	01-10-2023	38	No	No	No	No	No	No	No	No
47	S	1548231	CONTROL	26	Primigravida	3rd	12-02-2023	19-11-2023	38	No	No	No	No	Yes	No	No	No
48	Sk	10-11-4587	CONTROL	33	Multigravida	3rd	15-03-2023	22-12-2023	38	No	No	No	No	Yes	No	No	No
49	VA	14-10-5438	CONTROL	36	Multigravida	3rd	05-03-2023	12-12-2023	40	Yes	No	No	No	Yes	No	No	No
50	RM	25-01-1921	CONTROL	35	Multigravida	3rd	12-03-2023	19-12-2023	37	No	No	No	No	Yes	No	No	No

SR.No.	Bleeding Per Vaginum/ Spotting	Pallor	Icterus	Hepatomegaly	Ascitis	Increased BP readings	ICP	Hyperemesis Gravidarum	Acute Liver Failure	HELLP	Acute Fatty Liver of Pregnancy	Pre eclampsia	Acute on Chronic Liver Failure	Drug Induced Liver Injury	Hb	TLC	Platelet count	AST	ALT
1	No	No	No	No	No	Yes									9.7	12	317	26	28
2	Yes	No	No	No	No	No									11.5	12.3	376	44	51
3	No	No	No	No	No	No									11.3	8.27	136	55	78
4	No	No	No	No	No	No									11	9.08	350	36	56
5	No	No	No	No	No	No									12.3	7.7	168	57	59
6	No	No	No	No	No	No									13	8.9	150	28	40
7	No	No	No	No	No	No									12.5	11.8	272	30	42
8	No	No	No	No	No	No									3.5	11.9	268	81	48
9	No	No	No	No	No	No									11.3	9.4	140	16	13
10	No	No	No	No	No	No									11.7	21.1	169	21	15
11	No	No	No	No	No	No									12.3	9.6	229	76	56
12	No	No	No	No	No	No									10.9	11.83	175	56	67
13	No	No	No	No	No	No									11.3	6	182	51	67
14	No	No	No	No	No	No									9.9	11.31	164	35	29
15	No	No	No	No	No	No									12.6	11.3	274	31	25
16	No	No	No	No	No	No									8.8	11	237	15	12
17	No	No	No	No	No	No									12.5	9.6	165	26	23
18	No	No	No	No	No	No									12.1	6.9	116	23	31
19	No	No	No	No	No	No									8.7	9.7	192	22	26
20	No	No	No	No	No	No									12	5.9	122	36	40
21	No	No	No	No	No	No									11.3	8.6	267	47	48
22	No	No	No	No	No	No									13	11.5	157	14	11
23	No	No	No	No	No	No									10.5	6.2	110	20	24
24	No	No	No	No	No	No									10.4	11.7	230	13	11
25	No	No	No	No	No	No									12.2	8.3	150	25	8
26	No	No	No	No	No	No									9.5	11.9	268	81	48
27	No	No	No	No	No	No									11	15.9	196	43	36
28	No	No	No	No	No	No									13.4	11.7	243	27	43
29	No	No	No	No	No	No									13	11.3	271	28	14
30	No	No	No	No	No	No									10.2	10	265	53	31
31	No	No	No	No	No	No									10.5	8.8	152	20	8
32	No	No	No	No	No	No									11.4	12.4	144	35	28
33	No	No	No	No	No	No									8.3	8.1	179	30	32
34	No	No	No	No	No	No									12.8	12.4	199	61	49
35	No	No	No	No	No	No									11.2	9	250	37	30
36	No	No	No	No	No	No									10	8.4	184	25	15
37	No	No	No	No	No	No									13.4	10.6	257	30	22
38	No	No	No	No	No	No									11.8	11.5	104	50	93
39	No	No	No	No	No	No									12.9	9.7	303	45	35
40	No	No	No	No	No	No									11	8.3	200	42	43
41	No	No	No	No	No	No									10	7.5	120	17	18
42	No	No	No	No	No	No									11.6	7.5	213	20	13
43	No	Yes	No	No	No	No									5.2	12.9	277	10	14
44	Yes	No	No	No	No	No									13	12.3	298	14	13
45	No	No	No	No	No	No									12.1	10.5	174	21	17
46	Yes	No	No	No	No	No									10.6	11.7	155	17	19
47	No	No	No	No	No	No									11.5	5.9	160	25	23
48	No	No	No	No	No	No									12.2	10.8	208	30	18
49	No	No	No	No	No	No									11.9	15.4	202	26	13
50	No	No	No	No	No	No									11.8	13.1	176	19	12

SR.No.	ALP	Bile acids	Total Bilirubin	D. Bilirubin	Total Protein	S. Albumin	HIV	HAV	HEV	HBsAg	HCV	Dengue	Leptospirosis	Scrub typhus	Mode of delivery	Indications Of Cesarean Section	Fetal GENDER	Fetal Weight
1	78		0.2	0.06	6.4	3.55	NR	NA	NA	NR	NR	NA	NA	NA	LSCS		Male	2.08
2	396		1.46	1.3	6.3	3.41	NR	NA	NA	NR	NR	NA	NA	NA	Preterm NVD		Female	1.46
3	224		0.18	0.12	6.3	3.57	NR	NA	NA	NR	NR	NA	NA	NA	LSCS	Fetal distress	Male Female	1.8,2.06
4	194	15.1	0.21	0.08	7	3.83	NR	NR	NR	NR	NR	NA	NA	NA	LSCS	Previous LSCS	Female	2.6
5	287	5.2	0.06	0.03	6.1	3.13	NR	NA	NA	NR	NR	NA	NA	NA	Preterm NVD		Male	0.56
6	224		0.15	0.1	6.2	3.74	NR	NA	NA	NR	NR	NA	NA	NA	Term NVD		Female	2.6
7	153		0.35	0.06	7	3.78	NR	NA	NA	NR	NR	NA	NA	NA	LSCS	Fetal distress	Female	2.9
8	222		0.39	0.08	6.7	3.1	NR	NA	NA	NR	NR	NA	NA	NA	LSCS	Fetal distress	Male	3.2
9	105		0.49	0.06	6.5	3.2	NR	NA	NA	NR	NR	NA	NA	NA	LSCS	Fetal distress	Female	2.86
10	114		0.46	0.09	5.9	3.9	NR	NA	NA	NR	NR	NA	NA	NA	LSCS		Female	2.78
11	120		0.44	0.3	6.1	3.6	NR	NA	NA	NR	NR	NA	NA	NA	Term NVD		Male	3.69
12	155	16	0.14	0.04	7.1	3.17	NR	NA	NA	NR	NR	NA	NA	NA	LSCS	Fetal distress	Male	3
13	229		0.16	0.04	6.6	3.76	NR	NA	NA	NR	NR	NA	NA	NA	LSCS	Fetal distress	Male	3.4
14	110		0.15	0.02	6.1	3.4	NR	NA	NA	0	NR	NA	NA	NA	LSCS	Decreased Fetal movements	Male	2.8
15	120		0.9	0.4	7	4	NR	NA	NA	NR	NR	NA	NA	NA	Term NVD		Female	3.02
16	66		0.2	0.1	6.2	3.6	NR	NA	NA	NR	NR	NA	NA	NA	Term NVD		Female	3.2
17	111		0.3	0.09	6.8	3.4	NR	NA	NA	NR	NR	NA	NA	NA	Term NVD		Female	2.2
18	80		0.6	0.42	6	4	NR	NA	NA	NR	NR	NA	NA	NA	Term NVD		Male	4
19	72		0.72	0.33	5.8	3.9	NR	NA	NA	NR	NR	NA	NA	NA	Term NVD		Female	2.7
20	239		0.3	0.05	6.5	3.58	NR	NA	NA	NR	NR	NA	NA	NA	Term NVD		Female	3
21	160		0.6	0.11	5.1	3.9	NR	NA	NA	NR	NR	NA	NA	NA	Term NVD		Male	2.7
22	135		0.05	0.01	6.4	4	NR	NA	NA	NR	NR	NA	NA	NA	Term NVD		Male	3.1
23	80		0.44	0.13	5.9	4.1	NR	NA	NA	NR	NR	NA	NA	NA	Term NVD		Male	3.1
24	78		0.3	0.05	6	3.7	NR	NA	NA	NR	NR	NA	NA	NA	Term NVD		Male	3.2
25	168		0.61	0.14	6.2	3.3	NR	NA	NA	NR	NR	NA	NA	NA	LSCS	APH	Female	3.1
26	222		0.61	0.31	5.8	3.6	NR	NA	NA	NR	NR	NA	NA	NA	LSCS	Fetal distress	Male	3.2
27	112		0.11	0.09	6.1	3.9	NR	NA	NA	NR	NR	NA	NA	NA	Term NVD		Female	2.5
28	132	13.2	0.43	0.23	5.7	2.95	NR	NA	NA	NR	NR	NA	NA	NA	LSCS	Scar tenderness	Male	3.4
29	81		0.22	0.1	7	3.6	NR	NA	NA	NR	NR	NA	NA	NA	Term NVD		Female	2.6
30	68		0.12	0.08	5.82	3.11	NR	NA	NA	NR	NR	NA	NA	NA	LSCS		Male	2.5
31	125		0.16	0.09	6.8	3.8	NR	NA	NA	NR	NR	NA	NA	NA	LSCS	APH	Male	3
32	214		0.2	0.07	5.8	3.5	NR	NA	NA	NR	NR	NA	NA	NA	Term NVD		Male	2.9
33	78		0.6	0.2	6.5	3.5	NR	NA	NA	NR	NR	NA	NA	NA	Term NVD		Male	2.5
34	568		2.5	1.36	6.3	3.1	NR	NA	NA	NR	NR	NA	NA	NA	Term NVD		Male	2.7
35	86		0.33	0.16	5.9	3.6	NR	NA	NA	NR	NR	NA	NA	NA	Term NVD		Male	3
36	58		0.5	0.14	6	3.1	NR	NA	NA	NR	NR	NA	NA	NA	Assisted Vaginal Delivery		Male	3.5
37	72		0.88	0.5	5.5	3.4	NR	NA	NA	NR	NR	NA	NA	NA	Term NVD		Male	3.2
38	212		0.22	0.06	6.9	4.06	NR	NA	NA	NR	NR	NA	NA	NA	Assisted Vaginal Delivery		Male	2.3
39	78		0.8	0.61	5.8	4.1	NR	NA	NA	NR	NR	NA	NA	NA	Term NVD		Female	3
40	80		0.67	0.41	6.9	4	NR	NA	NA	NR	NR	NA	NA	NA	Assisted Vaginal Delivery		Male	3.6
41	128		0.2	0.11	5.9	3.39	NR	NA	NA	NR	NR	NA	NA	NA	Term NVD		Female	2.8
42	120		0.3	0.1	7	4.5	NR	NA	NA	NR	NR	NA	NA	NA	Term NVD		Male	3.3
43	70		0.22	0.09	6	3.4	NR	NA	NA	NR	NR	NA	NA	NA	Assisted Vaginal Delivery		Male	2.8
44	227		0.58	0.2	6.9	3.9	NR	NA	NA	NR	NR	NA	NA	NA	Term NVD		Female	3.4
45	220		0.69	0.41	6.5	3.5	NR	NA	NA	NR	NR	NA	NA	NA	Term NVD		Female	2.5
46	110		0.77	0.5	6	4.1	NR	NA	NA	NR	NR	NA	NA	NA	Term NVD		Male	2.3
47	124		0.3	0.02	6.2	4	NR	NA	NA	NR	NR	NA	NA	NA	Term NVD		Male	2.8
48	210		1.08	0.12	7.6	3.84	NR	NA	NA	NR	NR	NA	NA	NA	Term NVD		Male	3.1
49	114		0.6	0.14	6.3	3.2	NR	NA	NA	NR	NR	NA	NA	NA	Term NVD		Female	2.84
50	127		0.16	0.09	6.3	3.66	NR	NA	NA	NR	NR	NA	NA	NA	Term NVD		Female	2.7

SR.No.	Fetal Outcome	Fetal complications (If Any)	Maternal Outcome	Maternal Complications (If Any)	Disease
1	Normal Live Birth		Normal		
2	Preterm birth		Normal		
3	Normal Live Birth		Normal		
4	Normal Live Birth		Normal		
5	**Stillbirth**		Normal		
6	Normal Live Birth		Normal		
7	Normal Live Birth		Normal		
8	Normal Live Birth		Normal		
9	Normal Live Birth		Normal		
10	Normal Live Birth		Normal		
11	Normal Live Birth		Normal		
12	Normal Live Birth		Normal		
13	Normal Live Birth		Normal		
14	Normal Live Birth		Normal		
15	Normal Live Birth		Normal		
16	Normal Live Birth		Normal		
17	Normal Live Birth		Normal		
18	Normal Live Birth		Normal		
19	Normal Live Birth		Normal		
20	Normal Live Birth		Normal		
21	Normal Live Birth		Normal		
22	Normal Live Birth		Normal		
23	Normal Live Birth		Normal		
24	Normal Live Birth		Normal		
25	Normal Live Birth		Normal		
26	Normal Live Birth		Normal		
27	Normal Live Birth		Normal		
28	Normal Live Birth		Normal		
29	Normal Live Birth		Normal		
30	Normal Live Birth		Normal		
31	Normal Live Birth		Normal		
32	Normal Live Birth		Normal		
33	Normal Live Birth		Normal		
34	Normal Live Birth		Normal		
35	Normal Live Birth		Normal		
36	Normal Live Birth		Normal		
37	Normal Live Birth		Normal		
38	Intrauterine Death		Normal		
39	Normal Live Birth		Normal		
40	Normal Live Birth		Normal		
41	Normal Live Birth		Normal		
42	Normal Live Birth		Normal		
43	Normal Live Birth		Prolonged Hospital stay		
44	Normal Live Birth		Normal		
45	Normal Live Birth		Normal		
46	Normal Live Birth		Normal		
47	Normal Live Birth		Normal		
48	Normal Live Birth		Normal		
49	Normal Live Birth		Normal		
50	Normal Live Birth		Normal		

Baba Farid University
of Health Sciences
Sadiq Road
Faridkot-151 203 (Pb). INDIA.
Phone : 01639-256232,256236
Tele/Fax : 01639-256235
Email – Thesisbfuhs@gmail.com

(19)

No. BFUHS/2K23p-TH/ 13159 **Date** 27/12/23

The Principal
Dayanand Medical College & Hospital
Ludhiana

Subject:: Approval of Plan of Thesis.

This is to inform you that the Faculty of Medical Sciences in its meeting held on 25.09.2023 has approved the plan of thesis of **Arshdeep Kaur**, a student of your college for **MD General Medicine** along with the names of Supervisor/Co-Supervisor as under:-

Topic Plan of Thesis
To study the clinical profile of liver disease in pregnant females admitted in hospital & its effect on materno-fetal outcome .

Supervisor
Dr. Sandeep Puri
Professor
Dept. of Medicine

Co-Supervisor:-
Dr. Suman Puri
Prof. & Head (Unit-II),
Dept. of Obstetrics & Gynaecology,

Co-Supervisor:-
Dr. Omesh Goyal
Professor
Dept. of Gastroenterology

The student may submit the thesis in triplicate alongwith the plan in triplicate also, to the Controller of Examinations, BFUHS, Faridkot, at least six months before the commencement of theory examination, if he/she is otherwise eligible to take the examination. He /She may be directed to attach a photocopy of this letter with the forwarding letter while submitting thesis in the University along with the letter of approval of University for change of supervisor/Co-Supervisor, change of topic, if any.

Controller of Examinations

Note :

1. *If any discrepancy found regarding topic of thesis or Supervisor/Co-Supervisor, inform undersigned within one month after issue of this letter.*

TO STUDY THE CLINICAL PROFILE OF LIVER DISEASE IN PREGNANT FEMALES ADMITTED IN HOSPITAL & ITS EFFECT ON MATERNO-FETAL OUTCOME

THESIS PROPOSAL

FOR APPROVAL OF SUBJECT OF THESIS TO BE SUBMITTED IN

PARTIAL FULFILMENT OF THE REQUIREMENTS

FOR THE DEGREE OF

MD

In

GENERAL MEDICINE

OF THE

BABA FARID UNIVERSITY OF HEALTH SCIENCES

FARIDKOT

AUGUST, 2022

DR. ARSHDEEP KAUR

DEPARTMENT OF GENERAL MEDICINE
DAYANAND MEDICAL COLLEGE AND HOSPITAL
LUDHIANA

ABSTRACT OF PLAN OF THESIS

Title	TO STUDY THE CLINICAL PROFILE OF LIVER DISEASE IN PREGNANT FEMALES ADMITTED IN HOSPITAL & ITS EFFECT ON MATERNO-FETAL OUTCOME
For the degree of	MD General Medicine
Name of the candidate	Dr. Arshdeep Kaur
Supervisor	Dr. Sandeep Puri, Professor, Department of Medicine, DMCH, Ludhiana
Co-Supervisor	Dr. Suman Puri, Professor & Head (Unit II), Department of Obst & Gynae, DMCH, Ludhiana Dr. Omesh Goyal, Professor, Department of Gastroenterology, DMCH, Ludhiana
Institution	Dayanand Medical College and Hospital, Ludhiana
University	Baba Farid University of Health Sciences, Faridkot

Introduction: One of the least studied topics in the field of medicine is liver disease during pregnancy, which creates a challenge for both gynecologists and physicians. Approximately 3% of pregnant women are affected by some form of liver disease during pregnancy. Some of these conditions can be fatal for both the mother and child. Three types of liver disease need to be differentiated during pregnancy. One type is liver disease directly related to pregnancy, which occurs at a specific time during pregnancy. Another type is liver disease not related to pregnancy, which can occur at any time, such as viral- or drug-induced hepatitis. Furthermore, pregnancy can occur in women with pre-existing liver disease. it is essential that the clinicians are familiar with these disorders so they can respond promptly and appropriately in all of these situations, especially when emergency delivery is needed and must not be postponed.

Aim of Study:

- To study the clinical profile of liver disease in pregnant females admitted in hospital.
- To study and compare the Materno-fetal outcome among hospitalized pregnant females with or without liver disease

Materials & methods:

- Sample Size - 100 Cases of hospitalized pregnant females with liver disease as per inclusion criteria
- Sample Size- 50 Controls Of Hospitalized Pregnant Females Without Liver Disease
- Liver function tests will be studied and materno- fetal outcomes will be compared between two groups.
- Total number of pregnancies will be noted out of which pregnant females fulfilling the criteria will be included.

Study Type: Observational Prospective Study.

(Signature) Candidate	(Signature) Co-supervisor	(Signature) Co-supervisor	(Signature) Supervisor	(Signature) Principal

CERTIFICATE OF FACILITIES AVAILABLE

This is to certify that the facilities for work on the subject of thesis titled **"TO STUDY THE CLINICAL PROFILE OF LIVER DISEASE IN PREGNANT FEMALES ADMITTED IN HOSPITAL & ITS EFFECT ON MATERNO-FETAL OUTCOME"** exist in the Department of General Medicine at Dayanand Medical College and Hospital, Ludhiana and will be provided to the candidate. We will see that the data being included in the thesis are genuine and is collected by the candidate herself under our supervision and guidance.

Supervisor

Dr. Sandeep Puri
Professor
Department of General Medicine
DMCH, Ludhiana

Co-Supervisor's

Dr. Suman Puri
Professor & Head (Unit II)
Department of Obst & Gynae
DMCH, Ludhiana

Dr. Omesh Goyal
Professor
Department of Gastroenterology
DMCH, Ludhiana

Head of the Department

Dr. Dinesh Gupta
Professor & Head
Department of General Medicine
DMCH, Ludhiana

Place : Ludhiana

Date : 24/9/2022

APPROVAL PROFORMA
BY
RESEARCH & ETHICAL COMMITTEE
DAYANAND MEDICAL COLLEGE & HOSPITAL, LUDHIANA

Name of candidate	DR. ARSHDEEP KAUR
Department	GENERAL MEDICINE
Topic of Thesis	TO STUDY THE CLINICAL PROFILE OF LIVER DISEASE IN PREGNANT FEMALES ADMITTED IN HOSPITAL & ITS EFFECT ON MATERNO-FETAL OUTCOME
Likely date of appearing in PG examination	MAY/JUNE, 2024
Date of enrollment	MARCH 1, 2022
Name of Head of Department	DR. DINESH GUPTA
1) Supervisor **2) Co-Supervisor**	DR. SANDEEP PURI DR. SUMAN PURI DR. OMESH GOYAL
Signature of Members of Research Committee with Stamp	**Signature of Members of Ethics Committee with Stamp**
Dr. Harpreet Kaur (Professor & Head, Pathology)	Mr. R K Sharma (Legal Expert)
Dr. Jaspal Singh (Professor & Head, Surgery)	Dr. Ramit Mahajan (Associate Professor, Gastroenterology)
Dr. Dinesh Gupta (Professor & Head, Medicine)	Dr. Anu Sharma (Professor, Anatomy)
Dr. Sandeep Kaushal (Dean Academics & Professor & Head, Pharmacology)	Dr. Sandeep Kaushal (Dean Academics & Professor & Head, Pharmacology)
Dr. Gurpreet S Wander (Vice Principal)	Dr. Gurpreet S Wander (Vice Principal & Professor & Head, Cardiology)
	Dr. Gagandeep Singh (Secretary, Ethics Committee · Professor & Head, Neurology)

Approved: Yes / ~~No~~

Dr. Sandeep Puri
Chairperson
Research Committee
Chairperson
Research Committee

Approved: Yes / ~~No~~

Prof. Arvind Malhotra
Chairperson
Ethics Committee
Chairperson
Ethics Committee

BABA FARID UNIVERSITY OF HEALTH SCIENCES, FARIDKOT
PROFORMA FOR REGISTRATION OF SUBJECT FOR DISSERTATION

1	NAME OF THE CANDIDATE AND ADDRESS	DR. ARSHDEEP KAUR HOUSE NO.1780/2JR, KAPOOR ENCLAVE , JAGRAON , LUDHIANA
2	NAME OF THE INSTITUTE	DAYANAND MEDICAL COLLEGE AND HOSPITAL
3	COURSE OF STUDY AND SUBJECT	M.D. GENERAL MEDICINE
4	DATE OF ADMISSION OF COURSE	01/03/2022
5	TITLE OF THE TOPIC	TO STUDY THE CLINICAL PROFILE OF LIVER DISEASE IN PREGNANT FEMALES ADMITTED IN HOSPITAL & ITS EFFECT ON MATERNO-FETAL OUTCOME

6. BRIEF RESUME OF THE INTENDED WORK

6.1 NEED FOR THE STUDY

Liver dysfunction during pregnancy is multifactorial in origin and diagnosis is often challenging. The key to maternal and fetal wellbeing is an early diagnosis and appropriate management. Hepatic disorders complicate about 3% of all pregnancies and fall under various categories.

Firstly a heterogeneous group of liver disorders that are unique to pregnancy and occur in patients with a previously healthy liver. These include intrahepatic cholestasis of pregnancy, acute fatty liver of pregnancy and liver dysfunction associated with hyperemesis gravidarum and preeclampsia. These conditions remit spontaneously in puerperium.

Secondly, pregnancy may occur in background of a preexisting liver disease e.g. chronic viral hepatitis and cirrhosis liver.

Third category is of common viral diseases like acute viral hepatitis which may occur incidentally during pregnancy.

The fourth situation is that of disorders which are probably related to pregnancy e.g. biliary tract disease and Budd-Chiari Syndrome. Although still enigmatic, there have been recent interesting advances in understanding of these unique pregnancy related liver diseases. Hyperemesis gravidarum is an intractable, dehydrating vomiting in the first trimester of pregnancy; 50% of patients with this condition have liver dysfunction. Intrahepatic cholestasis of pregnancy has pruritus and elevated bile acids in the second half of pregnancy, accompanied by high levels of aminotransferases and mild jaundice. Maternal management is symptomatic with ursodeoxycholic acid; for the fetus, however, this is a high-risk pregnancy requiring close fetal monitoring and early delivery. Severe preeclampsia itself is the commonest cause of hepatic tenderness and liver dysfunction in pregnancy and 2-12% of cases are further complicated by hemolysis (H), elevated liver tests (EL), and low platelet count (LP)- the HELLP syndrome. Immediate

delivery is the only definitive therapy, but many maternal complications can occur, including abruptio placentae, renal failure, subcapsular hematomas, and hepatic rupture. Acute fatty liver of pregnancy is a sudden catastrophic illness occurring almost exclusively in the third trimester; microvesicular fatty infiltration of hepatocytes causes acute liver failure with coagulopathy and encephalopathy. Early diagnosis and immediate delivery are essential for maternal and fetal survival. Pregnancy causes very few alterations in the results of standard liver tests. The aminotransferases (AST and ALT), γ-glutamyltranspeptidase (GGTP), total bilirubin, and serum bile acid level remain within the normal range. Alkaline phosphatase rises modestly in the third trimester. The albumin level is lower than in nonpregnant women, while the cholesterol levels are higher. Thus, elevations in aminotransferases or GGTP signify pathology, and should prompt a search for disease. Making the correct diagnosis is of paramount importance, as failure to do so can result in morbidity or mortality for not only the mother, but also for her fetus. Identifying the aetiology and its effective management in a timely manner. A coordinated team approach that involves the primary care physician, obstetrician, hepatologist and neonatologist, is often required to promote good maternal and fetal outcome.

6.2 REVIEW OF LITERATURE

Pregnancy associated liver diseases are the most frequent cause of liver dysfunction in pregnancy, their prevalence is estimated to be 3%,[1] although variability by country and race/ethnicity is recognized, maternal mortality trends in the United States, assessed using death certificates, show an overall increase in deaths caused by liver disorders, from 1.25 to 8.80 per 1,000,000 live births from 1999–2001 to 2016–2018.[2] Liver disease during pregnancy is classified into two main categories: those related to pregnancy; and those non-related or unrelated. Liver disease unrelated to pregnancy can present *de novo* in pregnancy, or pregnancy can occur in women with pre-existing liver pathology. Pregnancy related liver diseases include intrahepatic cholestasis of pregnancy (ICP), acute fatty liver of pregnancy (AFLP) and, hemolysis, elevated liver enzymes and low platelets count (HELLP) syndrome. In addition, pre-eclampsia (PE) and hyperemesis gravidarum (HG) are frequently associated to liver abnormalities.[3-5] Non-related pregnancy hepatic diseases include acute and chronic viral hepatitis, cirrhosis with or without portal hypertension, biliary diseases such as gallstones and primary sclerosing cholangitis, vascular alterations such as Budd–Chiari syndrome (BCS), Wilson's disease, autoimmune liver diseases, metabolic disorders, drug induced hepatotoxicity and post liver transplantation state.[4,6-8] Amongst these, there is a wide spectrum of severity ranging from minor biochemical abnormalities to acute liver failure (ALF). Outcomes at the more severe end of the spectrum are difficult to predict and prognostic information is limited to small series.[9-11] Liver disorders may present themselves inconspicuously during gestation. Due to the physiological changes in the liver that occur during pregnancy, and huge variation in the spectrum of disease and presentation resulting in delays in diagnosis and appropriate management it is difficult to diagnose and manage a liver disease. Research and subsequent advances in medical care have resulted in improved but still not satisfactory maternal and fetal outcomes. Therefore a systematic approach is needed for its treatment and must include a detailed clinical history, examination, laboratory

analyses and radiographic evaluation. The clinical history should include previous pregnancy diseases and associated liver complications, intravenous drug use, transfusions, if pregnancies were taken to term, and oral contraceptive use. Also, the patient should be evaluated for clinical data that may suggest liver dysfunction such as nausea, vomiting, jaundice, generalized pruritus, abdominal pain, polyuria and polydipsia in the absence of other morbidities as chronic metabolic disease such as diabetes.[5,7,12-14]

The disorders are complex and patients benefit from multi-disciplinary input by experienced physicians in specialist centres. It is important to ensure that women of childbearing age have contraceptive advice that takes their liver disease into consideration, and to give them informed pre-pregnancy counselling. Once women with liver disease become pregnant, it is essential that they have rapid referral to specialist physicians with experience of managing hepatic disorders in pregnancy. Maternal and fetal outcomes are improving due to ongoing research, improved guidelines and our better understanding of preconception risk stratification, disease mechanisms and therapeutic options. to pregnancy that are present de novo, or are pre-existing chronic liver disease exacerbated by pregnancy.[6,7,13]

6.3 AIMS AND OBJECTIVES OF THE STUDY

- To study the clinical profile of liver disease in pregnant females admitted in hospital.
- To study and compare the Materno-fetal outcome among hospitalized pregnant females with or without liver disease

7. MATERIALS AND METHODS

A prospective study will be conducted in 100 cases of pregnant females with liver disease and 50 controls pregnant females with or without liver disease admitted in department of medicine and allied specialties in a tertiary care hospital, Dayanand Medical College and Hospital, Ludhiana during a 15 month period from 15th September, 2022 to 15th December, 2023

Liver function tests with be studied and compared between the two groups.

7.1 Source of Data:

Hospitalized pregnant females with liver disease admitted at medicine and other allied specialties at Dayanand Medical College and Hospital, Ludhiana

7.2 METHOD OF COLLECTION OF DATA

INCLUSION CRITERION (for cases) include:

1) Female patients older than 21 years of age, willing and able to sign consent
2) Pregnant
3) Liver enzymes > 2 times elevated

EXCLUSION CRITERION –

a. Non pregnant females
b. Age < 21 years
c. Not able to give consent
d. Liver enzymes < 2 times elevated
e. Any other chronic disease- Chronic kidney disease/Chronic obstructive airway disease/coronary artery disease, epilepsy

INCLUSION CRITERION (FOR CONTROLS) -

Pregnant females > 21 years of age with < 2 times elevated liver enzymes

Methodology

Duration of Study - 15-09-2022 TO 15-12-2023

Number of cases - 100

Number of controls - 50

OUTCOME

- **MATERNAL**
  - PRE ECLAMPSIA
  - PRETERM DELIVERY
  - DIC
  - PPH
  - INCREASED HOSPITAL STAY
  - ICU ADMISSION
  - MULTIORGAN FAILURE
  - MATERNAL MORTALITY
- **FETAL**
  - LIVE BIRTH
  - PRETERM DELIVERY
  - INTRAUTERINE DEATH
  - SPONTANEOUS ABORTION
  - MECONIUM STAINED LIQUOR
  - PREMATURE RUPTURE OF MEMBRANES
  - FETAL GROWTH RESTRICTION

STATISTICAL ANALYSIS:

Data will be described in terms of range; mean ±standard deviation (± SD), median, frequencies (number of cases) and relative frequencies (percentages) as appropriate. To determine whether the data is normally distributed, a Kolmogorov-Smirnov test will be used. Comparison of quantitative variables between the study groups will be done using Student t-test and Mann Whitney U test for independent samples for parametric and non-parametric data respectively. For comparing categorical data, Chi square ($\chi 2$) test will be performed and fisher exact test will be used when the expected frequency is less than

5. A probability value (p value) less than 0.05 will be considered statistically significant. All statistical calculations will be done using (Statistical Package for the Social Science) SPSS 21version (SPSS Inc., Chicago, IL, USA) statistical program for Microsoft Windows.

7.3 DOES THE STUDY REQUIRE ANY INVESTIGATIONS OR INTERVENTIONS TO BE CONDUCTED ON PATIENTS OR OTHER HUMANS OR ANIMALS? IF SO, PLEASE DESCRIBE BRIEFLY.

Yes; Routine Blood investigations like CBC, LFT, RFT along with specialised tests to access the disease severity and organ damage; Antenatal monitoring to know the fetal wellbeing.

7.4 HAS ETHICAL CLEARANCE BEEN OBTAINED FROM YOUR INSTITUTION IN CASE OF 7.3?

Attached herewith.

8. LIST OF REFERENCES

1. Ch'ng CL, Morgan M, Hainsworth I, Kingham JG. Prospective study of liver dysfunction in pregnancy in Southwest Wales. Gut 2002;51:876–80.
2. Joseph KS, Boutin A, Lisonkova S, Muraca GM, Razaz N, John S, et al. Maternal Mortality in the United States: Recent Trends, Current Status, and Future Considerations. Obstet Gynecol. 2021;137:763-71.
3. Allen AM, Kim WR, Larson JJ, Rosedahl JK, Yawn BP, McKeon K, et al. The Epidemiology of Liver Diseases Unique to Pregnancy in a US Community: A Population-Based Study. Clin Gastroenterol Hepatol. 2016;14:287-94.
4. Westbrook RH, Dusheiko G, Williamson C. Pregnancy and liver disease. J Hepatol. 2016;64:933-45
5. Goel KD, Jamwal A, Ramachandran KA, Balasubramanian CE. Pregnancy-related liver disorders. J Clin Exp Hepatol. 2014;4:151-62
6. Joshi D, James A, Quaglia A, Westbrook RH, Heneghan MA. Liver disease in pregnancy. Lancet. 2010;375:594-605
7. Mikolasevic T, Filipec-Kanizaj I, Jakopcic I, Majurec A, Brncic-Fischer, Sobocan A, et al. Liver disease during pregnancy: a challenging clinical issue. Med Sci Monit. 2018;24:4080-90.
8. Kamimura K, Abe H, Kawai H, Kamimura H, Kobayashi Y, Nomoto M, et al. Advances in understanding and treating liver diseases during pregnancy: A review. World J Gastroenterol. 2015;21:5183-90.
9. Shames DB, Fernandez LA, Sollinger HW. Liver Transplantation for HELLP Syndrome. Liver Transplantation 2005;11:224–8.
10. Ockner SA, Brunt EM, Cohn SM, Krul ES, Hanto DW, Peters MG. Fulminant hepatic failure caused by acute fatty liver of pregnancy treated by orthoptic liver transplantation. Hepatology. 1990;11:59–64.

11. Pereira SP, O'Donohue J, Wendon JA, Williams R. Maternal and perinatal outcome in severe pregnancy-related liver disease. Hepatology. 1997;26:1258–62.
12. Kelly C, Pericleous M. Pregnancy-associated liver disease: a curriculum-based review. Frontline Gastroenterol. 2018;9:170-4
13. Gomes CF, Sousa M, Lourenço I, Martins D, Torres J. Gastrointestinal diseases during pregnancy: what does the gastroenterologist need to know? Ann Gastroenterol. 2018;31:385-94.
14. Sharma AV, John S. Pregnancy, liver disease. StatPearls [Internet] StatPearls Publishing, Treasure Island (FL); 2018.

PROFORMA

Name _____ Age _____ S No _____

MRD No _____Husband/Father's name _____

Address _____

_____

Phone number _____

Education _____

Occupation _____

Consent: Yes / No

Smoker: Yes / No Alcohol: Yes / No

Date of Admission_____ Date of Discharge_____

LMP _____ EDD _____

Date of Diagnosis _____

Any other co-morbidity: DM/ HTN / TB / Others _____

Any pre-existing renal / cardiac / hepatic/pulmonary disease _____

Trimester 1st / 2nd / 3rd

Symptoms :

- Asymptomatic Yes/No
- Persistent Nausea/vomiting Yes/No
- Yellowish Discoloration Yes/No
- Itching on body Yes/No
- Headache Yes/No
- Visual symptoms Yes/No
- Difficulty in breathing Yes/No
- Right upper quadrant pain Yes/No
- Loss of appetite Yes/No
- Weight loss of >5% of pre-pregnancy body weight Yes/No
- Extreme fatigue Yes/No
- Dark colored urine Yes/No
- Decreaed Urine output Yes/No
- Spotting Yes/No
- Decreased fetal movements Yes/No

Signs :

- Pallor Yes/No
- Icterus Yes/No
- Hepatomegaly Yes/No
- Edema Yes/No
- Increased BP readings Yes/No
- Ascites Yes/No

Signature of Candidate **Signature of Supervisor**

GENERAL PHYSICAL EXAMINATION
BP
HR
Height
Weight
BMI

SYSTEMIC EXAMINATION
CENTRAL NERVOUS SYSTEM
CARDIOVASCULAR SYSTEM
RESPIRATORY SYSTEM
ABDOMEN

Signature of Candidate **Signature of Supervisor**

INVESTIGATIONS
Hb
TLC
Platelet count
Packed cell volume
PT/INR
ALP
AST
ALT
T.Bilirubin
D.Bilirubin
GGT
Total Protein
S. Albumin
Uric acid
Urine r/e
S. Creatinine
ESR
CRP (Optional)
ANA
Any other investigation if available

Signature of Candidate **Signature of Supervisor**

VIRAL MARKERS:
HIV
HAV
HEV
HBsAg
HCV
HSV
CMV
Others

ULTRA-SONOGRAPHY REPORT:

Signature of Candidate **Signature of Supervisor**

Final Diagnosis : _____

Mode of Delivery: _____

Fetal outcome :

Live birth
Abortion
Intrauterine death
Pre-term birth
Fetal Growth restriction

Maternal Outcome :

Normal
Multiorgan failure /Prolonged Hospital stay
Maternal Mortality

Signature of Candidate **Signature of Supervisor**

INFORMED PATIENT CONSENT

Date: _____

I…….D/o, W/o, S/o……..…… am willing to participate in the study **"TO STUDY THE CLINICAL PROFILE OF LIVER DISEASE IN PREGNANT FEMALES ADMITTED IN HOSPITAL & ITS EFFECT ON MATERNO-FETAL OUTCOME"** The procedure with its complications has been explained to me in local vernacular language. I have fully understood that no special tests and extra expenditure in any form, merely for the purpose of thesis is being carried out. I am free to opt out the study any time without assigning any reason.

……. ……
Signature of the Witness Signature of the patient

Name:

Signature of Candidate **Signature of Supervisor**

मरीज अनुमति फार्म

तिथि _____

मैं _____ पत्नी/पुत्र/पुत्री _____

निवासी _____

इस शोधकार्य "**TO STUDY THE CLINICAL PROFILE OF LIVER DISEASE IN PREGNANT FEMALES ADMITTED IN HOSPITAL & ITS EFFECT ON MATERNO-FETAL OUTCOME**" में भाग लेने के लिए सहमत हूँ। मैं इस शोधकार्य में भाग लेने के लिए अपनी स्वेच्छा से तैयार हूँ। मैं पुष्टि करता/करती हूँ की यह स्वीकृति देने के लिए मुझे कोई आर्थिक प्रलोभन नहीं दिया गया और न ही दबाव डाला गया है। मुझे इस शोधकार्य का उद्देश्य स्पष्ट कर दिया गया है और मुझे यह विश्वास है की यह विज्ञान और मानवता के लाभ के लिए है। मुझे इस शोधकार्य में होने वाले जोखिम के बारे में समझा दिया गया है और मेरी समझ में यह जोखिम बहुत मामुली है।

मैं इस शोधकार्य में भाग लेने के सम्बन्ध में किसी भी समय अपनी अस्वीकृति व्यक्त कर सकता/सकती हूँ और मेरे अस्वीकृति व्यक्त करने का मेरे इलाज पर कोई प्रभाव नहीं पड़ेगा।

_____	_____
गवाह के हस्ताक्षर	रोगी के हस्ताक्षर/अंगूठे का निशान
नाम:	नाम:

_____

अन्वेषक के हस्ताक्षर

_____

विद्यार्थी के हस्ताक्षर

ਮਰੀਜ਼ ਰਜ਼ਾਮੰਦੀ ਫਾਰਮ

ਮਿਤੀ_____

ਮੈ_____ਪਤਨੀ/ਪੁੱਤਰ/ਪੁੱਤਰੀ_____

ਵਾਸੀ _____

“TO STUDY THE CLINICAL PROFILE OF LIVER DISEASE IN PREGNANT FEMALES ADMITTED IN HOSPITAL & ITS EFFECT ON MATERNO-FETAL OUTCOME” ਤੇ ਚਲ ਰਹੀ ਖੋਜ ਵਿਚ ਹਿੱਸਾ ਲੈਣ ਲਈ ਰਜ਼ਾਮੰਦ ਹਾਂ। ਇਸ ਖੋਜ ਕੰਮ ਵਿਚ ਹਿੱਸਾ ਲੈਣ ਲਈ ਮੈ ਆਪਣੀ ਮਰਜ਼ੀ ਨਾਲ ਤਿਆਰ ਹਾਂ। ਮੈ ਇਹ ਦਸਣਾ ਚਾਹੁੰਦਾ ਹਾਂ/ ਚਾਹੁੰਦੀ ਹਾਂ ਕੇ ਇਹ ਰਜ਼ਾਮੰਦੀ ਦੇਣ ਵਾਸਤੇ ਮੈਨੂ ਕਿਸੇ ਵੀ ਤਰਾਂ ਦਾ ਲੋਭ-ਲਾਲਚ ਨਹੀ ਦਿੱਤਾ ਗਿਆ ਅਤੇ ਨਾ ਹੀ ਕੋਈ ਦਬਾ ਪਾਇਆ ਗਿਆ ਹੈ। ਮੈਨੂ ਇਸ ਖੋਜ ਦਾ ਮਨੋਰਥ ਸਮਝਾ ਦਿੱਤਾ ਗਿਆ ਹੈ ਅਤੇ ਮੈਨੂ ਇਹ ਭਰੋਸਾ ਦਵਾਇਆ ਗਿਆ ਹੈ ਕੇ ਇਹ ਖੋਜ ਵਿਗਿਆਨ ਅਤੇ ਮਾਨਵਤਾ ਦੀ ਭਲਾਈ ਲਈ ਹੈ। ਮੈਨੂ ਇਸ ਖੋਜ ਵਿਚ ਹੋਣ ਵਾਲੇ ਖਤਰੇ ਬਾਰੇ ਸਮਝਾ ਦਿੱਤਾ ਗਿਆ ਹੈ ਅਤੇ ਮੈਨੂ ਇਹ ਯਕੀਨ ਹੈ ਕੇ ਇਹ ਖਤਰਾ ਬਹੁਤ ਮਾਮੂਲੀ ਹੈ। ਮੈਨੂ ਇਸ ਖੋਜ ਕੰਮ ਵਿਚ ਹਿੱਸਾ ਲੈਣ ਦਾ ਢੰਗ – ਤਰੀਕਾ ਸਮਝਾ ਦਿੱਤਾ ਗਿਆ ਹੈ।

ਮੈ ਇਸ ਖੋਜ ਕੰਮ ਵਿਚ ਹਿੱਸਾ ਲੈਣ ਬਾਰੇ ਆਪਣੀ ਰਜ਼ਾਮੰਦੀ ਕਿਸੇ ਵੀ ਸਮੇ ਵਾਪਸ ਲੈ ਸਕਦਾ ਹਾਂ/ ਸਕਦੀ ਹਾਂ ਅਤੇ ਮੇਰੀ ਰਜ਼ਾਮੰਦੀ ਵਾਪਸ ਲੈਣ ਦਾ ਮੇਰੇ ਇਲਾਜ ਤੇ ਕੋਈ ਅਸਰ ਨਹੀ ਹੋਵੇਗਾ।

_____ _____

ਗਵਾਹ ਦੇ ਹਸਤਾਖਰ ਮਰੀਜ਼ ਦੇ ਹਸਤਾਖਰ

ਮਿਤੀ_____ ਮਿਤੀ_____

ਪਤਾ _____ ਪਤਾ _____

_____

ਨਿਰੀਖਕ ਦੇ ਹਸਤਾਖਰ

_____

ਸਿਖਿਆਰਥੀ ਦੇ ਹਸਤਾਖਰ